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Table of Contents

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ORIGINAL ARTICLES— PAGE.

"The Action of Radiation from Radium Needles on Nerves," by DAPHNE L. GOULSTON, B.Sc. .. 651

"Specific Desensitization in Asthma and Hay Fever," by CHARLES SUTHERLAND, M.B., M.R.C.P. 661

REPORTS OF CASES—

"Two Cases of Congenital Pancreatic Steatorrhœa with Infantilism," by MARGARET H. HARPER, M.B., Ch.M. 663

"Traumatic Partial Pneumothorax," by GUY GRIFFITHS, M.D. 664

REVIEWS—

Physical Signs in Chest Disease 665

LEADING ARTICLES—

Clinical Pathologists 667

CURRENT COMMENT—

Geographical Distribution of Rheumatic Fever .. 668

Emphysema Following Tonsillectomy 669

ABSTRACTS FROM CURRENT MEDICAL LITERATURE—

Physiology 670

Biological Chemistry 671

SPECIAL ARTICLES ON DIAGNOSIS— PAGE.

Nasal Sinusitis 672

BRITISH MEDICAL ASSOCIATION NEWS—

Scientific 673

OBITUARY—

Arthur Norris Wilkinson 676

CORRESPONDENCE—

Spreading Gangrenous Inflammation 676

Post-Graduate Education in Tasmania 677

CORRIGENDUM 677

PROCEEDINGS OF THE AUSTRALIAN MEDICAL BOARDS—

New South Wales 677

DIARY FOR THE MONTH 678

MEDICAL APPOINTMENTS VACANT, ETC. 678

MEDICAL APPOINTMENTS: IMPORTANT NOTICE 678

EDITORIAL NOTICES 678

THE ACTION OF RADIATION FROM RADIUM NEEDLES ON NERVES.[1]

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Sydney.

PART I.

THE information and figures set forth in this article are of a preliminary nature, subject to further additions as the investigation proceeds. The literature available on this subject is very scanty and vague. The gist of any articles found which dealt with the effect of radium on nerves is given in the following *résumé.*

Lazarus Barlow and Dunbar[(1)] experimented with whole nerve muscle preparations of frogs, using applicators holding seven milligrammes of radium bromide ($RaBr_2$). Using α rays, they concluded from their experiments which dealt with the minimal electric stimulation of nerve required to produce a contraction of muscles, that these α rays were beneficial to neuro-muscular irritability. They also found that β and γ rays after nine hours' radiation were ineffective.

Redfield and Forbes[(2)] radiated the nerve of a muscle preparation with β and γ radiations from a radium emanation tube containing 12 to 180 millicuries for periods from twelve to twenty-four hours. They found a disturbance of conductivity and irritability of the preparation, and also claim that fatty degeneration occurred in the nerve after their exposures.

Okada,[(3)] using ten milligrammes of radium bromide in a capsule, first shaved and then radiated

[1] This work was carried out under the control of the Cancer Research Committeee of the University of Sydney and with the aid of the Cancer Research and Treatment Fund.

the thighs of four rabbits directly over the sciatic nerve. A received twenty-four hours' radiation only. B, C and D received twenty-four hours' radiation and after a lapse of two to seventeen days another similar dose. The details of his experiments are very vague. After a period of thirty to forty days no histological change was observed in the sciatic nerve, although most intense cutaneous reactions were noted.

Scholtz[4] radiated several frogs and rabbits at the site of the sciatic nerve with twenty-five milligrammes of radium bromide in an india rubber capsule with a mica filter. The nerves, when dissected seven days later, appeared quite normal.

Dobrovolskaia-Zavadskaia,[5] investigating the action of radium element and radium emanation on tissues in close proximity to the sciatic nerve gave a table of details of (i) quantity of radiation, (ii) filtration by screens, (iii) duration of exposure (up to eleven months). They then gave a general statement that effects of the radium vary greatly according to the dose *et cetera*. Filtration protected the tissues in a marked fashion. The distance of the nerve from the focus was very important. Sensory function was more readily affected than motor. Trophic lesions did not correspond to the other disorders. Recovery was never observed. The continuity of the nerve trunk was maintained, but histological changes were evident and the tendency to regeneration was feeble. He deduced these from the following (I give radium figures only): The animal used was the rabbit, the nerve was the sciatic. The radium needle contained 1·99 milligrammes of radium and filtration was by platinum 0·5 millimetre, that is, the length of the active part of the radium needle contained 1·99 milligrammes and it was encased in a platinum covering 0·55 millimetre in thickness.

In Experiment A, after forty-eight days' radiation the rabbit survived for three months twenty days (the dose was 17·28 millicuries). In Experiment B, after nine months twelve days' radiation the rabbit survived for nine months twelve days (the dose was 103,000 millicuries).

There were no further details in this author's paper whose results were stated only in the general terms quoted above.

Cowell and Russ[6] state that Horowitz inserted one milligramme of radium bromide into nerve tissue and obtained atrophic changes in the axis cylinder and destruction of the myelin sheath, but unfortunately this reference is unprocurable and no details of duration of exposure are mentioned.

Scope of Investigation.

The series of experiments were so designed to try and formulate answers to the following questions:

1. Will γ radiation from radium element cause atrophy of the nerve fibre?

2. If so, what is the minimum time that must elapse before atrophic changes are observed?

3. If (1) occurs, what is the minimum dose necessary to produce atrophy?

Since in some cases two months were allowed to elapse before the animals were examined, these series of experiments had necessarily to be spread over a long period and have been in progress for over twelve months.

The distance of the radium needle from the tissue played a great part in the strength of the dose (*vide* "the law of the inverse squares"). Uniformity was gained by placing the needle as closely as possible in contact with the nerve in every case, as described in the operative procedure below.

Technique.

The animals were anæsthetized with ether, the fur was removed from the thigh area and an incision about one cubic centimetre in length was made through the skin on the posterior aspect of the thigh immediately above the sciatic nerve. The fibres of the *gluteus maximus* were separated for a length of about one centimetre to expose the upper portion of the sciatic nerve.

The radium needles were then introduced in such a manner that they lay over and in contact with and parallel to the sciatic nerve, the eye of the nerve being directed proximally.

One or two gut stitches were placed in the muscle and the skin incision was closed with silk. The operations were carried out under strict aseptic conditions.

The following strengths of radium needles were employed:

10 mgm. radium element, gold filter 0·5 mm. thickness.
5 mgm. radium element, gold filter 0·5 mm. thickness.
4 mgm. radium element, gold filter 0·5 mm. thickness.
3 mgm. radium element, platinum filter 0·5 mm. thickness.
2 mgm. radium element, platinum filter 0·5 mm. thickness.
1 mgm. radium element, platinum filter 0·5 mm. thickness.

The ten milligramme needle has an active length of two centimetres, whilst all the others have an active length of one centimetre.

After a certain period of time (*vide* tables) the animals were killed by an overdosage of ether. The whole sciatic nerve and its branches were dissected out and fixed in a solution of 1% osmic acid for two days, then washed and teased in a glycerine-water mixture. Parts of the teased nerve were mounted in a Farrant's solution and examined microscopically.

Experimental Findings.

It was noted that a considerable amount of fibrous tissue was formed around the site of radiation. The longer the radium was allowed to remain, the greater the amount of tissue formed.

An interesting fact concerning the blood supply was also noted. More than the ordinary number of blood vessels as compared with the normal side formed around the site of the radium and on removal of the needle intense hæmorrhage occurred, thus greatly impeding the experiments. This was found to occur especially with large doses.

The muscle at the site of radiation showed local signs of degeneration, in some places becoming quite gelatinous in constitution. Slides were made of some of these muscles and no being stained with hæmatoxylin and eosin showed considerable loss of structure of the muscle fibres.

Sections of skin stained with hæmatoxylin and eosin, taken from one animal whose sciatic nerve had been radiated with a three milligramme needle for six weeks, showed hypertrophy and keratosis of the epidermis. Epilation occurs rarely.

In general, whenever through the agency of the rays degeneration did occur, the following diagram will serve to represent what occurred.

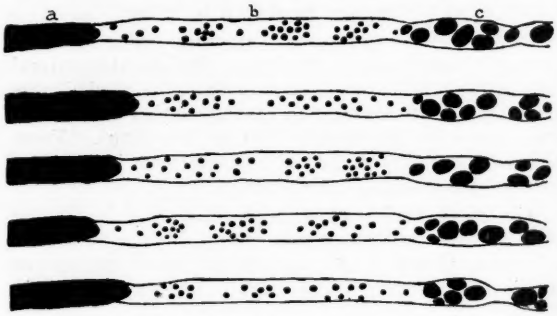


DIAGRAM I.

Illustrating the reaction of five nerve fibres to radiation from a radium needle. At *a* the portion of each nerve fibre situated centrally stains normally; while at *b* very minute globules indicating advanced atrophy are seen, and at *c* the portion of each fibre distal to *b* shows the much larger globules indicating early secondary degeneration.

Local atrophy occurred at the site of the radium element, while above it, that is, centrally, neither atrophy nor degeneration occurred. The local atrophy was limited in extent and definitely marked off in a sharp manner, as can be seen from Figures I and II.

Further, the local atrophied area was measured and for a one centimetre needle the segment was found to be three to four millimetres which is four to five nodes, whilst a two centimetre needle, that is 10 milligrammes, gave a length of atrophied nerve of nine to ten millimetres. There was intense local atrophy of the nerve fibres at the site of radiation, the myelin sheath breaking up into very minute droplets (*vide* Figures I and III). Below this atrophied area, that is, peripherally, Wallerian degeneration of the myelin sheath was shown (*vide* Figure I).

Table I records results showing that no changes are observed in the nerve for several days, even with considerable intensity of radiation.

"Experimental period" is the term used to express the time that was allowed to elapse between the insertion of the needle and the killing of the animal.

"Dosage" = $Q \times T$, Q being the quantity of radium and T the time.

"Secondary Wallerian degeneration" means the Wallerian degeneration below the radiated area which takes place as a result of the local atrophy at the site of the radium.

The highest dosage given was 160 milligramme-days and in each case the pig was killed immediately after radiation. In all these cases the nerve fibres appeared normal.

Rats were also used for experimental purposes and the same procedure adopted. Table II shows similar results to those recorded in Table I.

Table III records results in guinea-pigs in which local atrophy followed by secondary Wallerian degeneration occurred when the experimental period exceeded thirty-three days. On comparing this series of figures with the figures from Tables I and II, it will be seen that the dosages are approximately the same, but the time period is considerably longer.

TABLE I.

No.	Radium, Milligrammes.	Radiated Period in Days.	Dosage in Milligramme-Days.	Experimental Period in Days.	Result.
34	10	16	160	16	No signs of secondary Wallerian degeneration.
2	10	14	140	14	
7	10	13	130	13	
15	5	21	105	21	
16	10	13	130	13	
17	5	21	105	21	

TABLE II.

No.	Radium, Milligrammes.	Radiated Period in Days.	Dosage in Milligramme-Days.	Experimental Period in Days.	Result.
2	5	17	85	17	No signs of secondary Wallerian degeneration.
8	5	18	90	18	
9	4	18	72	18	
1	4	17	68	17	
5	3	19	57	17	
4	3	16	48	16	
11	3	15	45	15	
10	5	8	40	8	
3	3	11	33	11	
7	4	8	32	8	
6	3	7	21	7	

TABLE III.

No.	Radium, Milligrammes.	Radiated Period in Days.	Dosage in Milligramme-Days.	Experimental Period in Days.	Result.
33	5	33	165	33	Intense atrophy and secondary Wallerian degeneration.
31	3	56	168	56	
13	3	42	126	42	
18	3	47	141	47	
28	4	35	140	36	
26	3	36	108	36	

Smaller amounts of radium were left in contact with the nerve for a longer period. Thus the dosage, as indicated by milligramme-days, was approximately the same. Whereas in Table I twenty-one days was the longest time interval, in Table III intense local atrophy and Wallerian degeneration were seen. The action of the radium might have been initiated in the shorter time, but some period between twenty and thirty days must be necessary before the action can be microscopically seen.

A further series of experiments was designed to test this assumption. The results are recorded as follow in Table IV.

Rats were also used in a similar series of experiments and gave similar results recorded in Table V.

Even though in this series smaller dosages were used, the experimental period was prolonged for at least forty-four days. Local atrophy, secondary Wallerian degeneration and normal fibres were present. It was concluded then that the large dosages used in the other experiments give rise to physical and chemical changes, but that a latent period must elapse before these changes produce histological manifestations of the effects of radiations.

An attempt was then made to estimate this latent period. In some nerve fibres from an animal with an experimental period of twenty-nine days early Wallerian degeneration of approximately four to five days was seen. Hence the latent period was taken to be twenty-four days. From a consideration of the results in guinea-pigs with smaller and larger experimental periods, it would appear that approximately twenty-five days must pass before the changes could be identified. Table VI records the results of experiments in which insufficient radiation was employed, although long experimental periods followed the radiation.

The next series was as shown in Table VI.

In this series only small amounts were radiated over a small number of days. Before the animal was killed, however, the experimental period was prolonged. In specimens of this series no change was noted in the appearance of the fibres. From this it is concluded that the dosage was insufficient to produce histological response.

The next series of experiments was instituted using a three milligramme needle for a longer period of days, giving a dosage of at least twenty-one milligramme-days. The results are appended in Table VII.

From this it will be seen that local atrophy and secondary Wallerian degeneration occurred in many nerve fibres. Some normal fibres were also observed, a fact to which reference is made later. From this result it is inferred that it is the dosage of the radiation rather than the quantity of radium used which is of importance.

No signs of any attempt at regeneration of the nerve fibres have been observed in any of the specimens from this or any other series of experiments.

As will be seen from a comparison of Tables VI and VII, recording experiments in which a three milligramme radium needle was allowed to operate for two days and was then removed, the nerves exhibited none of the histological changes, even though the experiment was allowed to progress for

TABLE IV.

No.	Radium, Milligrammes.	Radiated Period in Days.	Dosage in Milligramme-Days.	Experimental Period in Days.	Result.
44	10	3	30	56	Local atrophy and secondary Wallerian degeneration in many but not all fibres and also normal fibres.
48	5	3	15	60	
50	4	7	28	60	
54	5	2	10	61	
43	3	3	9	44	

TABLE V.

No.	Radium, Milligrammes.	Radiated Period in Days.	Dosage in Milligramme-Days.	Experimental Period in Days.	Result.
74	3	7	21	33	Local atrophy and secondary Wallerian degeneration in many but not all nerve fibres and also normal fibres.
13	3	28	84	28	

ILLUSTRATIONS TO THE ARTICLE BY DAPHNE L. GOULSTON.

FIGURE I.

FIGURE II.

FIGURE III.

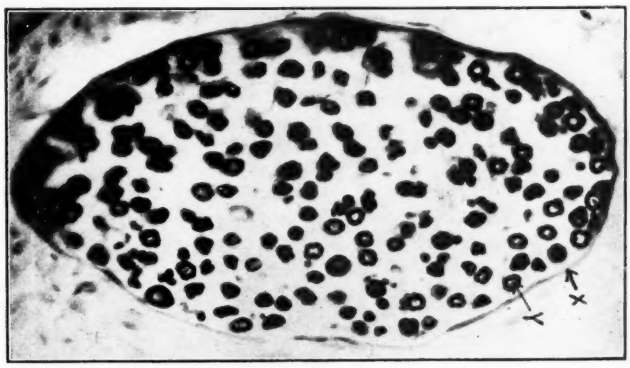


FIGURE IV.

ILLUSTRATIONS TO THE ARTICLE BY DAPHNE L. GOULSTON.

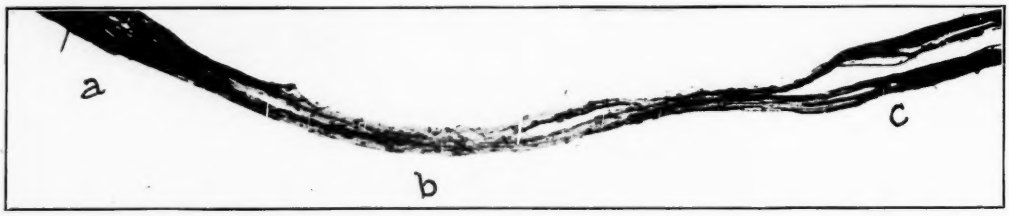


FIGURE V.

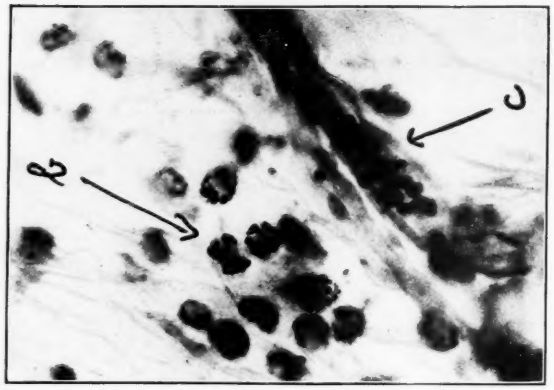


FIGURE VI.

FIGURE VII.

FIGURE VIII.

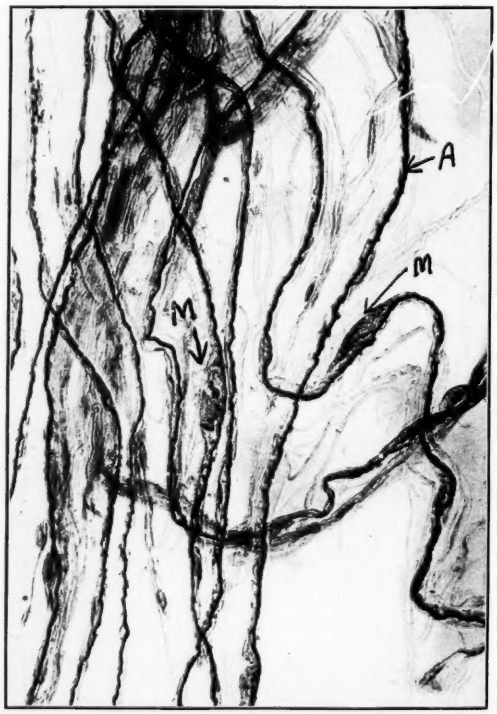


FIGURE IXA.

ILLUSTRATIONS TO THE ARTICLE BY DAPHNE L. GOULSTON.

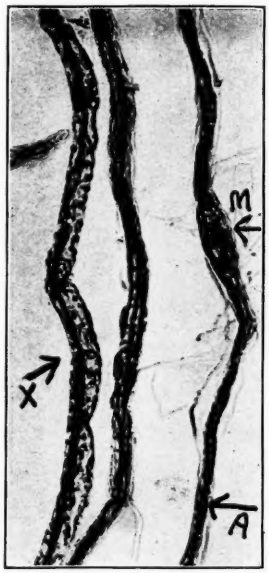


FIGURE IXB.

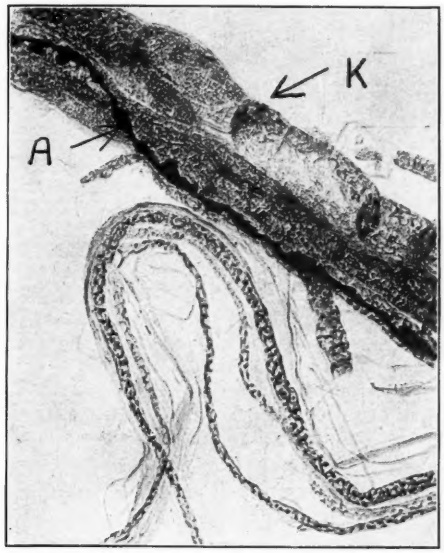


FIGURE X.

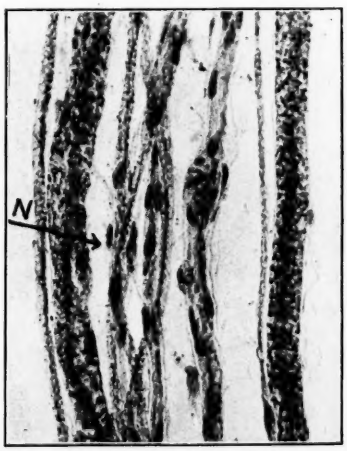


FIGURE XI.

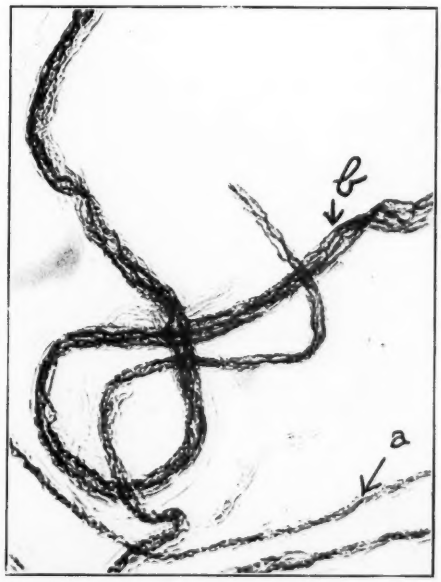


FIGURE XII.

ILLUSTRATIONS TO THE ARTICLE BY DAPHNE L. GOULSTON.

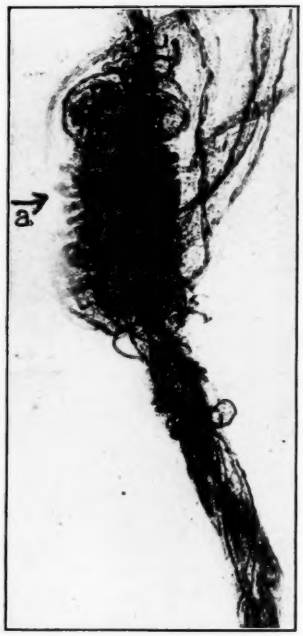


FIGURE XIIIA.

FIGURE XIIIB.

FIGURE XIV.

FIGURE XV.

TABLE VI.

No.	Radium, Milligrammes.	Radiated Period in Days.	Dosage in Milligramme-Days.	Experimental Period in Days.	Result.
55	3	2	6	61	Fibres normal.
56	3	2	6	57	
57	3	2	6	57	
60	2	3	6	63	
49	2	3	6	60	
51	1	7	7	60	

sixty-one days. The fibres appeared normal. When the same three milligramme needle was left for seven days and removed, even though the experimental period was shorter, local atrophy and secondary Wallerian degeneration in some fibres and also normal fibre were present. A comparison of the results of the experiments recorded in Tables III and IV shows that the atrophy and secondary Wallerian degeneration occur in all the nerve fibres when the period of radiation is between thirty-three and fifty-six days, while many fibres remain unaffected when the radiation is limited to seven days, although the period of observation may extend from forty-four to sixty-one days.

A cross section taken of a specimen which was radiated with a ten milligramme needle for three days, that is, receiving a dosage of thirty milligramme-days, showed complete degeneration of the nerves in two funiculi out of a total of eight funiculi. Some of these six funiculi, however, showed a few degenerated fibres. An intense dosage of 140 milligramme-days produced degeneration throughout the whole width of the cross section. This result is in part due to the different intensities of the radiations operating on the funiculi, due to their varied distances from the needles and in part due to a selective action on some nerve fibres lying together in the same funiculus. This selective action may be due to a difference in the chemical components of the individual nerve fibres (*vide* Figure IV).

To demonstrate the total degeneration, an animal, pig number 28, which had been radiated with a four milligramme needle for thirty-five days, was anæsthetized with "Amytal" injections. The nerve was severed above the site of radiation. The distal end of the nerve showed complete lack of conductivity and irritability on electrical stimulation. A control experiment on the other sciatic nerve produced the usual contraction.

On comparing Table VII and III it will be seen that the large dosages used in the experiments mentioned in Table III were markedly reduced in the experiments recorded in Table VII. The experimental period was about the same. Normal fibres were present in the series recorded in Table VII, but were absent in that recorded in Table III. In Table VII the experimental period is just over the latent period. Normal fibres were still present in the nerves of the animals corresponding to the experiments mentioned in Table VII. On a comparison of the experiments recorded in Tables IV and VII, it will be seen that smaller dosage was used in the experiments mentioned in Table IV, though the experimental period was longer. Normal fibres were present, even though the latent period was well passed over. Hence it is concluded that the length of the period of radiation rather than the duration of the experimental period determines the presence or absence of normal fibres. Experiments on guinea-pigs were then initiated to determine the minimum dosage necessary to bring about the changes in the tissue. These experiments are still in progress, but Table VIII is appended containing records of two additional experiments to those already mentioned in Tables IV and VI.

TABLE VII.

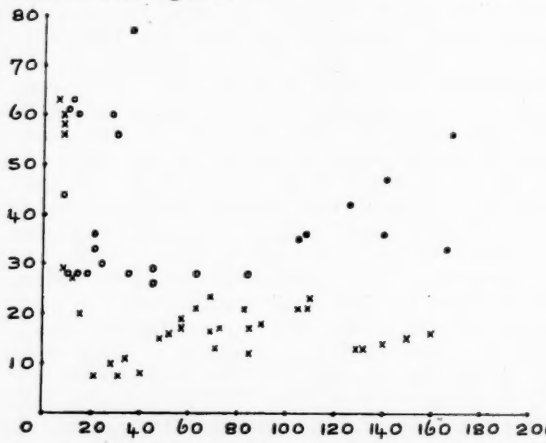
No.	Radium, Milligrammes.	Radiated Period in Days.	Dosage in Milligramme-Days.	Experimental Period in Days.	Result.
73	3	7	21	36	Local atrophy, secondary Wallerian degeneration and normal fibres.
38	3	13	45	26	
39	3	13	45	26	
40	3	15	45	29	
36	3	21	63	28	
37	3	21	63	28	

TABLE VIII.

No.	Radium, Milligrammes.	Radiated Period in Days.	Dosage in Milligramme-Days.	Experimental Period in Days.	Result.
54	5	2	10	61	Local atrophy and secondary Wallerian degeneration in some fibres and also normal fibres.
58	4	3	12	63	

On comparing Table VIII with Tables IV and VI it will be seen that a dosage of at least ten milligramme-days is necessary to produce visible microscopical changes. These experiments, however, are still in progress, but it appears that a dosage of approximately ten milligramme-days is necessary.

The teased specimens from the experimental animals recorded in Table VIII were most interesting. Some fibres showed the first apparent reaction to radium. A marked disintegration of the myelin sheath into small fatty droplets was found at the site of radiation with an apparently normal fibre both proximal and distal to the site of disintegration (*vide* Figure V). This is the earliest stage in local atrophy of a nerve fibre. It occurs prior to the onset of secondary Wallerian degeneration below the site of radiation. Nerves exposed to a threshold dose of radiation contained not only fibres showing secondary Wallerian degeneration, but also many fibres which showed a local atrophy only, although the experimental period was as long as sixty-three days. This fact suggested that with these minimal dosages the myelin sheaths alone were affected while the axones remained intact. This subject is being further investigated.



Graph showing "latent period" and the effect of dosage. The ordinate represents dosage in milligramme days, the abscissa represents experimental period in days. The cross represents normal nerve fibres, the black dot represents degenerate nerve fibres and the circle denotes the presence of both normal and degenerate nerve fibres.

Attention may be directed to the observation that normal fibres are seen frequently lying among the affected nerve fibres, not only in the region of local atrophy at the site of radiation, but also among the fibres showing secondary Wallerian degeneration. Some of these fibres lie in portion of the nerves close to the needle and others (usually more numerous) towards the opposite side of the nerve. Even when the local atrophy has progressed considerably, several normal fibres may traverse the greatly altered region.

Again, in branches of the nerve in which most of the fibres show secondary Wallerian degeneration, a few normal fibres may be seen. Such normal fibres are so numerous that no accidental intermingling of fibres during the preparation of the specimen could account for the result. It seems more probable that these differences depend on slightly different chemical composition which can readily lead to different degrees of absorption of the radiations.

Other interesting points discovered during the course of the experiments were as follow.

On staining portions of the nerves with iron-hæmatoxylin, it was seen that the cells of the normal neurilemma sheath stained very well and were quite regular. In the part of the nerve which had been radiated, however, the nuclei of these cells did not take up the stain at all well, the outlines were most irregular and the granules were not as distinct as in the ordinary cell.

Another interesting point noted was that, lying in the fibrous tissue of the epineurium, were large cells which took up the osmic acid stain very markedly. These cells were discovered to be leucocytes which had ingested numerous fatty droplets of the disintegrated myelin (*vide* Figure VI). Such leucocytes occurred at the site of the local atrophy, due to the radiation and in the portion of the nerve undergoing secondary Wallerian degeneration.

Radiation Energy.

The radium needles employed in the experiments in this paper may be represented as shown in Diagram II.

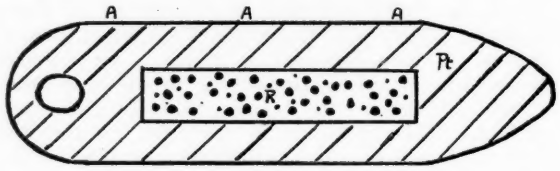


DIAGRAM II.

Illustrating the construction of the radium needles employed in the experiments. The needle is composed of platinum (*Pt.*), has a total length of two centimetres and an external diameter of 1·6 millimetres. An eye is situated at one end and the other end is a blunt point. The shaft of the needle is hollow and the radium chamber (*R*) so formed has a length of one centimetre and a wall of 0·5 millimetre of platinum.

Let three orthogonal axes, x, y, z, be drawn and let a small area, A, in the yz plane with its centre in the axis of x be taken. Suppose a platinum needle, containing one milligramme of radium element and having an active length of ten millimetres, an external diameter of 1·8 millimetres and a wall thickness of 0·5 millimetre, be placed along the axis of z. Assuming a line distribution of radium in the needle, a rough estimate of the amount of energy of any one type (γ, primary β, secondary β) falling on A can be made.

In the calculations A is taken equal to $\frac{1}{10}$ square millimetre. In the first column of each of the Tables IX, X, XI, XII and XIII the distances of A from the surface of the needle are given. The corresponding ray energies expressed in gramme calories per hour appear in the second column.

The γ rays penetrating the platinum walls of the needle give rise to secondary β rays. An approximation to the energy of these β rays can be obtained

by assuming a mean wave length for the primary γ rays. In Table XIII the first number in the second column gives the energy for a γ ray of 19 *X* units, the second number for a γ ray of 36 *X* units.

TABLE IX.

Axis of x passes through centre of needle, primary γ ray energies.
(0·5% is absorbed in passing through one millimetre of water.)

Position of Area *A* in Millimetres from the Surface.	Primary γ Ray Energy in Gramme Calories per Hour.
0	$1·2\times10^{-5}$
1	$0·6\times10^{-5}$
2	$0·4\times10^{-5}$
3	$0·2\times10^{-5}$
4	$0·2\times10^{-5}$
5	$0·1\times10^{-5}$

TABLE X.

Axis of x passes through centre of needle, primary β ray energies (same conditions).

Position of Area *A* in Millimetres from the Surface.	Primary β Ray Energy in Gramme Calories per Hour.
0	$3·3\times10^{-8}$
1	$1·6\times10^{-8}$
2	$1·0\times10^{-8}$
3	$0·7\times10^{-8}$
4	$0·5\times10^{-8}$
5	$0·4\times10^{-8}$

TABLE XI.

Axis of x passes through the end of needle, primary γ ray energy.

Position of Area *A* in Millimetres from the Surface.	Primary γ Ray Energy in Gramme Calories per Hour.
0	$0·6\times10^{-5}$
1	$0·3\times10^{-5}$
2	$0·2\times10^{-5}$
3	$0·1\times10^{-5}$
4	$0·1\times10^{-5}$
5	$0·1\times10^{-5}$

TABLE XII.

Axis of x passes through end of needle, primary β ray energies (same conditions).

Position of Area *A* in Millimetres from the Surface.	Primary β Ray Energy in Gramme Calories per Hour.
0	$1·7\times10^{-8}$
1	$0·8\times10^{-8}$
2	$0·5\times10^{-8}$
3	$0·3\times10^{-8}$
4	$0·3\times10^{-8}$
5	$0·3\times10^{-8}$

TABLE XIII.

Secondary β ray energies arising from primary γ rays, axis of x passes through centre of needle.
(About 60% absorbed in passing through one millimetre of water.)

Distance from Surface in Millimetres.	Gramme Calories per Hour.	*X* Units.
0	$5·6\times10^{-8}$	19
0	$3·8\times10^{-8}$	36

The energy radiated from either end of the active portion of the needle is only about half that radiated from the centre as illustrated in the foregoing tables.

From the tables it would appear that the amount of γ energy falling on one square millimetre surface of nerve immediately opposite the centre of the radium needle containing three milligrammes of radio-active element is $(1·2\times10^{-5}\times10\times3)$, that is, $3·6\times10^{-4}$ gramme calories per hour.

On the assumption that the nerve is two millimetres thick and that the γ rays are for practical purposes not absorbed in passing through two millimetres of nerve, the amount of γ energy leaving one square millimetre of nerve directly opposite the square millimetre of entry will be $(0·4\times10^{-8}\times10\times3)$, that is, $1·2\times10^{-4}$, a little less than one-third the amount entering on the opposite side of the nerve. A comparison of these amounts of energy with those found active in producing changes by Dr. Moppett and Mr. Love,[7] namely, $1·87\times10^{-8}$ gramme calories per hour on one square centimetre shows that these amounts are of the order of 2,000,000 times greater.

With similar assumptions concerning the β rays derived from the radium, the amount of β energy falling on a surface of one square millimetre immediately opposite the centre of a three milligramme radium needle will be $(3·3\times3\times10^{-7})$ and the amount of this energy leaving one square centimetre of nerve directly opposite the square millimetre of entry will be $(1·0\times3\times10^{-7})$, that is, $3·0\times10^{-7}$. Again, comparison of these amounts with the figure of Dr. Moppett and Mr. Love mentioned above shows these amounts to be of the order of 5,000 times greater.

During the minimal radiation period of eighty hours the amount of energy falling on a surface of the nerve of one square millimetre area will be $2·8\times10^{-2}$ gramme calories from the primary β rays.

The amount of energy derived from the secondary β rays which arise from the primary γ rays, radiated from a three milligramme radium needle is estimated at $(4\times10^{-7}\times3)$, that is, $1·2\times10^{-6}$ gramme calories per hour on one square millimetre. These larger differences in the amounts of effective energy, as found by Dr. Moppett and myself, are probably due to a difference in the chemical constituents of the allantoic membrane and sciatic nerve, and a difference in the wave lengths of the radiation employed.

Summary.

1. Two effects follow the application of γ rays from radium to nerves. These are a local atrophy at the site of radiation and secondary Wallerian degeneration in the nerve fibres below the atrophied region. The local atrophy is shown by disintegration of the myelin into droplets with subsequent gradual disappearance of the fatty droplets. The axis cylinder appears to be destroyed somewhat later than the splitting up of the myelin. The nuclei of the neurilemma become less distinct. The secondary Wallerian degeneration appears to be typical in its characters.

2. A certain period of from twenty to thirty days must elapse before any change can be noted in the nerve. The changes seen after this period elapsed are local atrophy at the site of radiation together with secondary Wallerian degeneration in the same nerve fibre peripherally, the central end remaining normal.

3. Radiations from three milligrammes of radium applied for a distance of one centimetre along the sciatic nerve of rats and guinea-pigs for three to ten days give rise to local atrophy and secondary Wallerian degeneration, but all the nerve fibres are not affected, normal fibres being still present. When the needle is applied for over thirty-five days, it produces local atrophy and secondary Wallerian degeneration in all the fibres.

5. The length of the atrophic segment measures three to four millimetres or nine to ten millimetres, according to the active length of the needle, either one or two centimetres respectively.

6. There is a minimal dosage below which no histological changes are induced in the nerve. This minimal dose is about ten milligramme-days.

7. There were no signs of regeneration in any of these nerves, even up to sixty-three days.

PART II.

Following on the previous experiments, in which the effect of radiation on the myelin sheath was particularly noted, further experiments to note the effect of radiation on the axone were instituted.

Technique.

A technique similar to that employed before was used, except that the nerve stained according to Cajal's silver nitrate method which consisted of fixation of the nerve in 95% alcohol for twenty-four hours, impregnation with a 1·5% silver nitrate solution in an incubator at 37° to 40° C. for six days, after which the specimen was washed and placed in a reducing liquid for twenty-four hours. This reducing liquid was composed of pyrogallic acid, 1 gramme; formalin, five cubic centimetres; water, 100 cubic centimetres.

The nerve was then kept in 75% alcohol and later teased in a 50% glycerine solution and examined microscopically. Non-radiated normal axones stain a pale reddish brown by this method, but radiated axones take up the stain much more deeply, staining a deep mahogany colour (*vide* Figure VII).

Experimental Findings.

The myelin sheath also took up some of this stain and the changes noted in the previous section could be followed and correlated with the changes in the axone.

Experiments were proceeded with to find out the effect of small doses (radiated for a small period) and left over approximately the latent period which in previous experiments was discovered to be necessary before changes could be seen. Table XIV is appended.

It was seen on microscopical examination of these specimens that local atrophy at the site of radiation of both the myelin and the axones had occurred.

Secondary Wallerian degeneration and normal fibres were also noted. A considerable amount of young fibrous tissue was found when teasing the nerve (*vide* Figure VIII).

As shown in Table XV small doses were given as before.

These, however, showed no change, except that the axone took on a deeper tinge than non-radiated fibres. The preceding tables were those of small dosages and small experimental periods. Experiments were then undertaken in which the animals were left for long experimental periods, as can be seen from Table XVI.

TABLE XIV.

Threshold Dosages—Silver Nitrate Staining.

No.	Radium, Milligrammes.	Dosage in Milligramme-Days.	Experimental Period in Days.	Result.
N 2	3	18	28	Local atrophy.
N 3	2	12	28	Secondary Wallerian degeneration and normal fibres.
N 10	4	24	30	Considerable amount of young fibrous tissue.

TABLE XV.

No.	Radium, Milligrammes.	Dosage in Milligramme-Days.	Experimental Period in Days.	Result.
N 4	1	12	28	Axones practically normal.
N 6	10	10	28	

TABLE XVI.
Threshold Dosages—Long Experimental Periods.

No.	Radium, Milligrammes.	Dosage in Milligramme-Days.	Experimental Period in Days.	Result.
68	4	20	122	(a) Degeneration of myelin at site of radiation and slight extension of myelin degeneration peripherally with normal axones.
77	5	15	106	
75	3	21	111	
62	10	10	124	
80	5	15	161	
79	4	12	169	(b) Normal fibres.
81	3	9	119	(c) An occasional secondary degenerated fibre.
65	10	10	168	

In the experiments marked with an asterisk the animals used were rats and in all the remaining experiments guinea-pigs were used. The nerve from rat number 80 was fixed in formal-alcohol-acetic and then stained with hæmatoxylin and eosin. Microscopical examination revealed great proliferation of nuclei of cells of Schwann at the site of radiation, as well as a few leucocytes.

In this it was seen that there occurred degeneration of the myelin sheath at the site of radiation with normal axones. This degeneration extended peripherally from the radiated area for a distance of about 0·5 centimetre. Figure IX shows this. At odd intervals what appears to be a nucleus occurs, but this is really an accumulation of globules of the degenerated myelin sheath; in these were seen normal axones with occasionally a secondary degenerated fibre.

Figure X showed an early stage in the reaction of the axone to radiation. Some nerve fibres appeared to have undergone some metamorphosis, the axone having an irregularly swollen outline. The skeleton outlines of other nerve fibres were seen where the axone had degenerated and had been absorbed.

In a section taken from rat number 80 a great proliferation of nuclei of the cells of Schwann at the site of radiation with a few leucocytes also was noted (*vide* Figure XI).

Following on the preceding experiments, subminimal doses were given to two animals, which were then allowed a long experimental period.

These showed practically no change in the axone, only intensifying of its staining.

Thus, so far, in the range of experiments with axones it was noted that with threshold doses degeneration occurred in the myelin sheath only, whilst no change occurred in the axones. This, even though the effect was observed six months after the radiation. There were only one or two cases over this long period in which any suggestion of regeneration or stimulative effects of the rays was noted. This was seen as a fibrillation of the axone, as shown in Figure XII. Another sign of regeneration was seen as a nervous spool in one case only, Figure XIII. There is also the possibility that the nerve fibres, mentioned in Table XVI, showing normal axones with fatty accumulations, are in reality regenerated nerve fibres. This matter will be investigated further.

To study the effect of radiation when the soft secondary rays were eliminated, experiments were undertaken with radium needles inserted into rubber tubing of varying thickness tied at both ends with sterilized silk. The results are set out in Table XVIII.

All specimens were stained with silver nitrate. N14 compared with other numbers in Table XVIII showed normal fibres. Since the intensity of radiation varies inversely as the square of the distance from its source, the needle was left in for a longer period to compensate for the lowering of its power due to the interpolation of the rubber screen.

As will be seen by referring to Table XVIII, N17 showed commencement of metamorphosis of the axone of some fibres at the site of radiation. Secondary Wallerian degeneration had not yet taken place.

In N15 similar results to the above were obtained. In this specimen, however, secondary Wallerian degeneration had taken place (*vide* Figures XIV and XV). Normal fibres were also present.

In N18B, even though a large dose over a small period was given, the fibres appeared normal. This again showed that degeneration due to radiation did not occur before the latent period had elapsed.

Another interesting experiment was the study of the "cross fire" effect of two radium needles in the nerve. Table XIX is appended.

The cross fire effect from a dosage of 48 milligramme-days produced local atrophy of both axone and myelin sheath and secondary Wallerian degeneration. In fact, no normal fibres were observed. When compared with Tables IV, V and

TABLE XVII.
Subminimal Dosages—Long Experimental Period.

No.	Radium, Milligrammes.	Dosage in Milligramme-Days.	Experimental Period in Days.	Result.
63	5	5	124	Practically normal.
64	3	3	172	Only reaction was an intensifying of staining at site of radiation.

TABLE XVIII.
Effect of Screened Radiation.

No.	Radium, Milligrammes.	Rubber Thickness, Millimetres.	Dosage in Milligramme-Days.	Experimental Period in Days.	Result.
N 14	3	1·0	18	32	Normal.
N 17	3	1·0	33	26	Commencement of metamorphosis of axone at site of radiation of some fibres. No secondary Wallerian degeneration.
N 15	5	1·0	35	28	Secondary Wallerian degeneration and normal fibres.
N 18B	10	1·0	150	15	Normal.

TABLE XIX.
Screened Radiation—Effect of Cross Fire.

No.	Radium Milligrammes.	Filtration Rubber, Millimetres.	Separate Dosage in Milligramme-Days.	Total Dosage in Milligramme-Days.	Experimental Period in Days.	Result.
N 29	3	0·5	24	48	27	Local atrophy and secondary Wallerian degeneration. No normal fibres observed.

VII, this is very interesting, as even with a dosage of 84 milligramme-days, normal fibres were observed, whereas none was seen in the rubber filtered "cross fire" radiation from a smaller dosage.

Summary.

1. With small doses of radiation degeneration of the myelin sheath only is observed, using three separate methods of staining, namely, silver nitrate, osmic acid and hæmatoxylin. No degeneration of the axone was observed even though the experimental period was prolonged to six months.

2. With sufficient dosage and experimental period degeneration of the axone does occur, local atrophy and secondary Wallerian degeneration again taking place.

3. Degeneration of the axone can be brought about by primary β and γ rays only, the soft secondary rays having been eliminated.

4. No statement can be made at present concerning the regenerative effect of radiation.

5. Great proliferation of the nuclei of the neurilemma sheath occurs at the site of radiation.

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LEGENDS TO ILLUSTRATIONS.

FIGURE I.

Microphotograph of a part of a nerve fibre which was radiated around *b*; *a* = nerve fibre situated central to the site of atrophy, stains normally with osmic acid; *b* shows advanced degeneration of the myelin sheath at the site of radiation; and *c* shows commencement of degeneration of the sheath distal to the site radium needle.

FIGURE II.

Microphotograph of two nerve fibres stained with osmic acid, showing at *x* the sharp transition from the normal to the degenerate myelin sheath at the site of radiation, *b*. The specimen, taken from sciatic nerve of guinea-pig number 40, was radiated with a three milligramme radium needle for fifteen days and the animal killed fourteen days later.

FIGURE III.

Microphotograph of a part of the atrophic segment at the site of radiation of a nerve radiated for eleven days with a three milligramme needle and allowed an experimental period of sixty-three days. The nerve was taken from guinea-pig number 28 and stained with osmic acid.

FIGURE IV.

Microphotograph of a transverse section of a funiculus of a nerve stained with osmic acid, showing scattered distribution of many degenerated nerve fibres. *y* = normal fibre. *x* = degenerate fibres containing globule of fat. Specimen from pig number 44. Nerve radiated with ten milligramme needle for three days. Experimental period, fifty-six days.

FIGURE V.

Microphotograph of some nerve fibres, stained with osmic acid, from the sciatic nerve of guinea-pig number 58. The nerve had been radiated around *b* with a four milligramme radium needle for three days, and the animal killed sixty days later. *b* fibres show sparsely stained atrophic segments with apparently normal nerve fibres above and below at *a* and *c*.

FIGURE VI.

Microphotograph showing fibrous tissue, infiltrated with phagocytes (*b*) containing fat globules, and a capillary with corpuscles in the lumen. Fibrous tissue taken from sciatic nerve of pig number 72, which was radiated with a five milligramme needle for seven days and allowed an experimental period of thirty-six days.

FIGURE VII.

Microphotograph of some nerve fibres central to the site of radiation. *A* = axone stained normally with silver nitrate. Guinea-pig number 29. Nerve cross radiated with two three milligramme needles for eight days. Experimental period, twenty-seven days.

FIGURE VIII.

Microphotograph of newly formed fibrous tissue obtained when teasing a nerve at the site of radiation. *N* = nuclei of cells. Nerve radiated with a two milligramme needle for six days. Silver nitrate staining. Pig number N3. Experimental period, twenty-eight days.

FIGURE IXA.

Microphotograph of some nerve fibres with normal axones (*A*) and the remains of degenerated myelin sheaths (*M*). Silver nitrate staining. Nerve radiated with a ten milligramme needle for one day. Guinea-pig number 62. Experimental period of 124 days.

FIGURE IXB.

Microphotograph of some nerve fibres with normal axones (*A*) and the remains of degenerated myelin sheaths (*M*). One normal nerve fibre is seen at *X*. Silver nitrate staining. Nerve radiated with a ten milligramme needle for one day. Guinea-pig number 62. Experimental period of 124 days.

FIGURE X.

Microphotograph showing several "skeleton" nerve fibres (*K*) and one metamorphosed axone (*A*). The nerve of the rat number 81 was radiated with a three milligramme needle for three days. Experimental period, 119 days. Silver nitrate staining.

FIGURE XI.

Microphotograph of nerve fibres stained with hæmatoxylin and eosin to show the great proliferation of the nuclei (*N*) of the neurilemma sheath. Nerve radiated with a three milligramme needle for five days. Rat number 80. Experimental period, 161 days.

FIGURE XII.

Microphotograph of a few nerve fibres stained with silver nitrate. *a* = normal axone, *b* = fibrillated axone. The nerve was radiated with a ten milligramme needle for one day. Experimental period, 124 days. Pig number 62.

FIGURE XIIIA.

Microphotograph showing some nerve fibres entwined in a *cul-de-sac* to form the nervous spool *a*. Nerve was radiated with a three milligramme needle for seven days. Pig number 75. Allowed experimental period of 111 days. Silver nitrate staining.

FIGURE XIIIB.

Microphotograph showing several swollen and deeply stained (metamorphosed) axones. The nerve of guinea-pig number 18B was radiated with a ten milligramme needle, screened with one millimetre rubber for fifteen days. Experimental period fifteen days. Silver nitrate staining.

FIGURE XIV.

Microphotograph of some nerve fibres showing secondary degeneration of axones below the site of radiation. *a* = fragments of axone stained with silver nitrate. The nerve was radiated through a screen of one millimetre of rubber with a five milligramme needle for seven days. Guinea-pig number N15. Experimental period, twenty-eight days.

FIGURE XV.

Microphotograph showing some nerve fibres degenerated at the site of radiation. Silver nitrate staining. The nerve was radiated with a five milligramme needle screened with one millimetre of rubber for seven days. Pig number 15. Experimental period, twenty-eight days.

SPECIFIC DESENSITIZATION IN ASTHMA AND HAY FEVER.

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TREATMENT of hay fever and asthma by desensitization is becoming so widely used that it seems that a brief summary of the specific methods of lessening hypersensitiveness should be useful.

This method was no doubt suggested when anti-anaphylaxis was discovered. If a guinea-pig is sensitized by injecting a small quantity of serum subcutaneously, it becomes hypersensitive, so that intravenous injection of a larger dose a few weeks later causes fatal anaphylactic shock. If before this second dose we give a few small doses of serum subcutaneously, the animal becomes "desensitized" and no serious results follow the intravenous injection. Apart from this, it has long been known that tolerance for poisons such as morphine can be enormously increased by a course of sublethal doses.

When a patient is found by skin tests to be hypersensitive to some pollen or epithelial product, we attempt to lessen his sensitiveness (and increase his tolerance) by injecting an extract of the substance in question in a series of increasing doses. At present extracts prepared according to Coca's method in buffered saline solution with 0·5% phenol are probably the most satisfactory available, and the following remarks on dosage refer particularly to these extracts. (In Australia they are obtainable in suitable dilutions from the Commonwealth Serum Laboratories, Melbourne, N.2.) For convenience a series of dilutions is used, usually labelled *A*, *B*, *C* and *D* strengths. *A* is the weakest, *B* is ten times stronger, *C* one hundred times and *D* one thousand times stronger than the *A* strength.

The *A* strength may be too weak or too strong for commencing treatment. To test this, inject a minute drop (about 0·01 cubic centimetre) intradermally on the upper arm. If, after twelve minutes, there is a very small itching wheal surrounded by slight redness, that is the correct strength. If there is no reaction at all, probably it is too weak. If there is a large reaction, it is too strong and a solution ten times weaker should be used. Another method of testing is to put four small scratches on the arm and rub a drop of *A* strength into one, *B* into the next, and so on. Treatment should be commenced with the solution which just fails to produce a wheal after ten or fifteen minutes.

Having found a suitable strength, give 0·1 cubic centimetre of this as a first dose and double the dose at each visit. Thus, as a rule, *A* strength is found suitable and the doses are: 0·1 cubic centimetre *A* strength, 0·2 cubic centimetre *A* strength, 0·4 cubic centimetre *A* strength, 0·8 cubic centimetre *A* strength.

Then take *B* strength, which is ten times stronger, and give similar increases: 0·1, 0·2, 0·4, 0·8 cubic centimetre of *B* strength.

In giving a course of treatment each dose requires rather careful consideration. On the one hand we have the risk of serious upset if too large a dose is given, and on the other hand treatment is liable to be hopelessly protracted if we are too cautious with the increases.

If the arm is inspected twenty minutes after an injection, it will be noticed that, as the doses get larger, each gives a local reaction. This consists of itching, redness and swelling, and sometimes wheals form on the skin. When injections produce marked local reactions it is unsafe to increase doses rapidly. Thus, if 0·2 cubic centimetre of the *C* strength produced an area of redness and swelling 2·5 or 5·0 centimetres (one or two inches) in diameter, one would not double the dose, but probably 0·3 cubic centimetre would be safe. Thereafter one must be guided chiefly by the extent of the local reaction and by the patient's observations. No rule can be given regarding the maximum dose. Of course, with very sensitive patients the optimum dose is likely to be much less than in less sensitive ones. The optimum dose may be less than the maximum for which there is tolerance, but as far as we can tell the maximum dose is the optimum also. Usually with pollens one cannot exceed 0·2 or 0·3 cubic centimetre of the *D* strength solution, but some take more and some much less than this. As a rule, if a certain dose is repeated every few days, there is progressively less and less reaction after each. But when very large doses are reached, one may find that tolerance, as judged by the local reaction, does not seem to increase at all, and it is wise to take this as the maximum.

Interval Between Doses.

We have very little data as to how long it takes to assimilate each dose. With smaller doses it seems safe to give one every day, but with larger ones the local reaction at the site of injection may take two or three days to disappear, and it seems wise to allow this to subside completely before giving more.

When what seems to be the optimum dose is reached, it may be repeated about once a week for a few weeks. Often it may be possible to stop treatment completely, but sometimes it is found necessary to give an occasional dose to maintain freedom from symptoms. There seems to be a distinct risk in giving large doses at intervals greater than a few weeks. If a patient was given, say, 0·4 cubic centimetre of *D* strength three months before, it would probably be wiser to give a "miniature course," say, 0·05, 0·15, 0·3 and 0·4 cubic centimetre of the *D* strength if he required further treatment. With a new patient a typical scheme of treatment would be:

A strength.—
0·1 cubic centimetre.
0·2 cubic centimetre.
0·4 cubic centimetre.
0·8 cubic centimetre.
(Interval between injections, 1 day in each instance.)

B strength.—
0·1 cubic centimetre.
0·2 cubic centimetre.
0·4 cubic centimetre.
0·8 cubic centimetre.
(Interval between injections, 2 days in each instance.)

C strength.—
0·1 cubic centimetre.
0·2 cubic centimetre.
0·4 cubic centimetre.
0·8 cubic centimetre.
(Interval between injections, 3 days in each instance.)

D strength.—
0·1 cubic centimetre.
0·2 cubic centimetre.
0·2 cubic centimetre.
0·25 cubic centimetre.
0·3 cubic centimetre.
0·4 cubic centimetre.
0·4 cubic centimetre.
0·45 cubic centimetre.
(Interval between injections, 4 to 7 days in each instance.)

This scheme should be regarded only as a rough guide, as each patient presents different problems. With rather sensitive persons it will usually be necessary to go more slowly through the *C* strength, and with the *D* strength much more conservative increases may be necessary. If time is very limited, it is possible sometimes safely to give doses of *A* and *B* strengths at intervals of only a few hours, so that *D* strength may be reached in seven days. But this method requires special vigilance and a fair amount of experience.

Standardization of Extracts.

It should be clearly recognized that there is no satisfactory method of standardization. Extracts are usually labelled as containing so many "units" per cubic centimetre. It was thought at one time that potency was proportional to the nitrogen content, but this is not always so. If a patient could tolerate, say, 0·3 cubic centimetre of *D* strength rye grass pollen extract (Batch 60), it would be highly dangerous to inject 0·3 cubic centimetre of *D* strength (Batch 70) which might, in fact, be ten times more potent. The only safe way is to keep to the same "batch" throughout the course.

Route for Injection.

As a rule extracts are injected subcutaneously into the upper and outer part of the arm. There are some reasons for thinking that intradermal injection of extracts gives better results and I frequently use this method, especially towards the end of a course. Usually it is impossible to inject more than 0·3 cubic centimetre intradermally at any one spot, so that where using *D* strength one may have to use two or three punctures for each dose. Of course, with doses in the weaker strengths, instead of giving, say, 0·8 cubic centimetre of *B* strength, one would give 0·08 cubic centimetre of *C* strength to avoid bulk.

Duration of Immunity.

For hay fever it is usually necessary to give treatment before each "hay fever season" for at least three years. Results are, of course, much better if treatment can be practically completed before the grasses begin to pollinate. During the season it is wise to give a dose every two or three weeks to maintain the tolerance. If asthma patients begin to show symptoms again eight or nine months after a course finishes, they will require a short supplementary course and subsequently should have a group of four or five injections every three months. Frequently the skin sensitiveness is appreciably lessened after a course and it is not uncommon to find that sensitiveness has disappeared completely.

Symptoms of Overdose: Constitutional Reactions.

Sometimes, if too large a dose is given, constitutional symptoms may appear. As a rule they are felt within a few minutes of the injection, but occasionally an hour or more elapses.

The regions affected vary greatly. A hay fever patient may get sudden acute irritation in the eyes and nose, or an asthmatic, quite well before the injection, may be seized with acute asthma; or perhaps there is simply slight tightness in the chest and substernal itching. Rhinorrhœa, generalized urticaria, œdema of the pharynx and congestion of the conjunctivæ are other symptoms. At the site of injection as a rule there is a large spreading wheal surrounded by erythema and small wheals frequently appear in the surrounding skin. Erythema may spread up the course of the lymphatics and the lymph glands in the axilla swell. In rare cases there is very little local reaction. With these overdoses the blood pressure falls, the pulse becomes very frequent and there may be dangerous collapse.

Treatment of Overdose.

Of course, every possible care should be taken to avoid overdosing. A large local reaction should always indicate caution in subsequent increases. The patient should wait for half an hour after each injection and adrenalin should always be handy. But, even with these precautions, one occasionally reaches the limit of tolerance without suspecting it.

If constitutional symptoms follow a dose, 0·3 to 0·6 mil (five to ten minims) of adrenalin (1 : 1,000) injected subcutaneously will usually check them. If after seven or eight minutes symptoms are not abating, it may be wise to repeat the dose of adrenalin. A tourniquet placed on the limb above the site of injection of the protein extract will help to check further absorption. Some authorities suggest in desperate cases intravenous administration of adrenalin, but its effects by this route are always very unpleasant and probably dangerous. Theoretically, atropine should be very useful. If there is circulatory failure, Cooke recommends strophanthin which, he says, acts better if no atropine has been given.

Reports of Cases.

TWO CASES OF CONGENITAL PANCREATIC STEATORRHŒA WITH INFANTILISM.

By MARGARET H. HARPER, M.B., Ch.M. (Sydney),
Honorary Physician, Royal Alexandra Hospital for Children.

PANCREATIC steatorrhœa in children has been reported by various authors and the cases may be divided into four groups:

1. Congenital steatorrhœa without infantilism. In these cases the patient from birth passes oily stools which solidify on cooling. In the cases reported by Garrod and Hurtley[1] and by Miller and Perkins[2] the passage of such stools continued apparently without interfering in any way with growth or nutrition. Apart from the steatorrhœa there was nothing to suggest disease of the pancreas in either case. Garrod suggests "that in the subjects of this anomaly some factor of the pancreatic secretion is wanting, an hypothesis which brings this condition into line with other known errors of metabolism."

2. Acquired pancreatic disease, such as may be associated with chronic enteritis.

3. Pancreatic infantilism with onset at puberty, such as the cases reported by Byron Bramwell.[3]

4. Congenital pancreatic infantilism. The two cases reported here fall into this group. Clarke and Hadfield[4] report a case of this type in which death occurred from bronchopneumonia at the age of four years and four months. The child, a girl, had the facial appearance and weight of a child of two and the height of a child of three years of age. In this case the liver was much enlarged. She had passed fatty stools from birth. At the *post mortem* examination it was found that the pancreas was represented by fat and a portion of normal gland tissue estimated to be about one-twentieth of the normal amount.

Case I.

Baby A., a female, was the fifth child of its mother. There was nothing of importance in the family history. The mother had been well during this pregnancy and the confinement was normal. The infant's birth weight was 3·7 kilograms (eight and a quarter pounds) and when she was first seen at the age of five months she weighed 3·78 kilograms (eight pounds six and a half ounces). Her height was 55·6 centimetres (twenty-two and a quarter inches), her head circumference was normal for her age, but her chest and abdominal measurements were those of an infant two months old. She had marked muscular hypotonia, and X ray examination of her bones revealed osteoporosis. Her stools were pale, lustrous with a rancid odour. The passage of oily material which solidified on cooling, accompanied the motion and occurred independently. The mother stated that oily stools had been present from birth. The motions were rather frequent, from four to five a day, and were sometimes green and slimy. The examination of the stools by Dr. Eva Shipton gave the following result: Total fat in dried fæces, 75·6%. Of this fat 53·8% was split and 46·2% was neutral fat. On a diet rich in protein and low in fat with a moderate amount of carbohydrate the infant gained weight and did fairly well. At seven months she weighed 4·3 kilograms (nine pounds thirteen ounces). She returned to her home in the country, where she continued to gain weight slowly. She died when about eleven months old of bronchopneumonia. No *post mortem* examination was obtained.

Case II.

Baby B., female, was the second child of its mother. The first child had died soon after birth from some congenital malformation the nature of which was not clear from the history. This infant weighed 2·7 kilograms (six pounds) at birth. She was first seen at the age of six and a half weeks. She was nearly 0·45 kilogram (one pound) under her birth weight and was small and miserable looking. The complaint made by her mother was that the infant's

stools were frequent, green and large, and that she was losing weight. She was breast fed, but was not receiving sufficient from her mother. On inspection the stools were found to be bright green in colour with little mucus. The mother's milk contained over 4% of fat. As the condition of the stools suggested an inability to deal fully with the fat in her diet, the baby was given supplementary feedings of dried skimmed milk. On this *régime* she improved, gained weight and the stools became a yellow paste. These were still rather frequent, but small in quantity. In a little over a fortnight the baby had regained her birth weight and appeared to be making normal progress. She returned to her home in the country and reports of fair progress were received, although the motions continued to be rather frequent and occasionally greenish in colour. She was seen again at the age of twelve months, when she was recovering from an attack of diarrhœa and vomiting. She was very small, showing general infantilism. Her height was 63·75 centimetres (twenty-five and a half inches) and her weight 5·7 kilograms (twelve pounds fourteen ounces), being about the height and weight of an infant of five or six months old. Her head was normal in size, contrasting oddly with her small body. She was plump, but not fat, and was beginning to walk with support and to say a few words. At this time her motions were large, pale, smooth, with a lustrous appearance and a rancid odour. They were passed four or five times in the twenty-four hours, mostly at night. The examination of the stools by Dr. Eva Shipton showed that the total fat of the dried fæces was 60%. Of this fat 76·6% was split and 23·4% was unsplit. Although the amount of unsplit fat in this case falls just within the normal limits as given by G. A. Harrison, it is considerably in excess of that usually found in cœliac disease (11% to 14% of fæcal fat is usually unsplit in the latter disorder). At no time was oil which solidified on cooling, seen in the evacuations of this infant, such as was present in the previous case. On a high protein, fat-free diet the child improved and gained weight for a few weeks. She then developed bronchopneumonia to which she succumbed.

Post Mortem Report.

I am indebted to Dr. F. Tidswell, Director of the Department of Pathology of the Royal Alexandra Hospital for Children, for the following report on the *post mortem* examination of this second child.

The body was emaciated, but otherwise normal in external appearance. In the thorax the lungs were crepitant anteriorly, but had several consolidated areas in both bases posteriorly. Microscopically these showed the usual pneumonic exudate with purulent material in the bronchi. This bronchopneumonia was apparently the cause of death. The heart and valves were normal and the *foramen ovale* was closed.

The abdomen was not swollen. The stomach was normal. No duodenal obstruction was found. No inflammatory signs were found in any part of the intestine and sections from various regions were normal histologically. Some of the mesenteric glands were prominent, but pale, and microscopically normal. Spleen, kidneys and suprarenals were macroscopically and microscopically normal. The liver showed congestion, cloudy swelling and moderate fatty degeneration. Portal areas and bile ducts were normal. There were manifest changes in the pancreas which was rather small and not of uniform character. One part of it was vaguely glandular, the other dense and fibrous. Microscopically the glandular part showed pancreatic tissue, but with much fibrous thickening of the trabecular and interlobular partitions. The widely separated alveoli were distorted and nearly all distended with secretion and their lining cells were flattened to a cuboidal shape. The ducts also were distended and their cells flattened. There were no definite islets, but Langerhans cells seemed to be represented by loose groups of epithelium-like cells. The fibrous part was a mixture of fibrous, fatty, smooth muscular and nerve tissue complicated beyond lucid description, but representing some embryonal inclusion in the pancreatic development. The condition of the pancreas suggests defective and obstructed external secretion interfering with proper fat (and other?) digestion and so accounting for the clinical observations.

References.

(1) A. E. Garrod and W. H. Hurtley: "Congenital Family Steatorrhœa," *The Quarterly Journal of Medicine*, 1912-1913, Volume VI, page 242.

(2) R. Miller and H. Perkins: "Congenital Steatorrhœa," *The Quarterly Journal of Medicine*, 1920-1921, Volume XIV, page 1.

(3) Byron Bramwell: "Clinical Studies," 1908, Volume VI, page 175.

(4) C. Clarke and G. Hadfield: "Congenital Pancreatic Disease with Infantilism," *The Quarterly Journal of Medicine*, 1923-1924, Volume XVII, page 358.

TRAUMATIC PARTIAL PNEUMOTHORAX.[1]

By GUY GRIFFITHS, M.D. (Sydney),
Senior Honorary Physician, Royal North Shore Hospital of Sydney.

A.C., A MAN of thirty years of age, was admitted to the Royal North Shore Hospital of Sydney on March 12, 1930, under my care, complaining of cough, vomiting, abdominal pain and illness for two weeks.

His history was that he had been wounded in France in 1918; a ball had entered his chest through the upper part of the sternum and passed out through the right scapula close to the shoulder joint. He had been desperately ill for some months and had had an operation in the lower and outer part of the back of the right chest to let out matter. Thereafter he had gradually improved until about sixteen months after the wound he had been discharged with a disability estimated at 40%, for which he still drew pension. For several years he had been bright and active and despite some stiffness at the right shoulder, earned his living as a carter, jumping up and down from his cart scores of times a day. About a fortnight ago he had epigastric pain and jaundice which grew worse till on March 10 he could work no longer, suffering as he did from pain and violent coughing which produced vomiting of foul-smelling and foul-tasting pus. As the vomiting gave ease he sometimes induced further attacks by pressing his finger down his throat, turning over on his left side and lowering his head so as to aid the discharge of pus.

His pulse was 100, his temperature 39·7° C. (103·6° F.) and his respirations 25 per minute.

He bore a very small pale scar in the upper part of his *gladiolus sterni* and a large, irregular, depressed scar behind his right shoulder, beneath which could be felt an irregularly united scapula. Lower down over the seventh and eighth ribs was the depressed scar suggesting an operation for empyema. His right arm was smaller and rather weaker than the left. He could not extend it above the level of the shoulder. There were no definite abdominal symptoms.

The respiration was a little distressed, with cough and copious purulent sputum up to 150 or 180 cubic centimetres (five or six ounces) per day. The right side of the chest moved poorly, the percussion note was greatly diminished all over, the breath sounds were faint and almost tubular towards the right base, and higher up were bronchovesicular with numerous râles. Towards the right base a friction rub could be heard.

The urine was clear, alkaline, of specific gravity 1016 and did not show either albumin or sugar.

He was treated with a mixture containing iron, opium and a little strychnine and sulphate of magnesia, with inhalation of creosote and carbolic acid from a perforated zinc mask and with counter-irritants to the epigastrium. He was kept on the veranda of the ward and given light diet with extra eggs and milk. He was encouraged to bend down out of bed towards the floor so as to aid his cough to expel his sputum.

[1] This patient was demonstrated at a meeting of the New South Wales Branch of the British Medical Association on June 12, 1930.

In three days his temperature fell to 37·8° C. (100° F.) and in five days to normal, and so continued. At the same time the dulness at the right base lessened and after free expectoration was temporarily replaced by resonance far out at the right pulmonary base.

I had at first contemplated using antistreptococcic serum, but as he defervesced so quickly and as no streptococci were found in his sputum and no tubercle bacilli, but only *Micrococcus catarrhalis*, the antiserum was not required.

A skiagram showed mottling suggesting a recent infection of the right lung and also a partial pyopneumothorax with enormous thickening of the pleura over the adjacent lung. The appearances are shown diagrammatically in the accompanying figure.

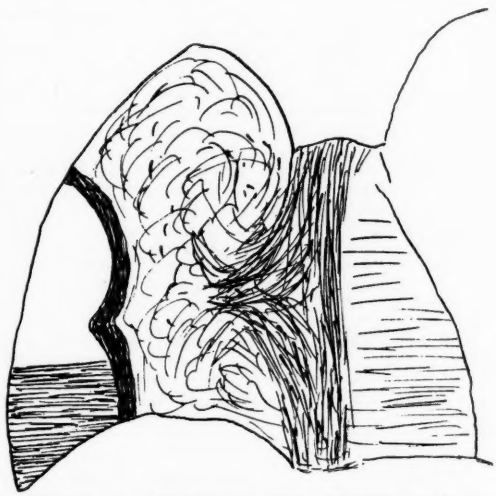

Diagram of skiagram of lung, showing partial pneumothorax with enormously thickened pleura covering the uncollapsed part of the right lung.

The patient convalesced rapidly and on March 18 his leucocyte count amounted only to 10,600 per cubic millimetre of peripheral blood with 73% polymorphonuclear cells, 25% lymphocytes and 2% eosinophile cells. On April 6 he was discharged to the Thomas Walker Convalescent Hospital. He continued to make good progress and two or three weeks later returned to duty.

On June 12 he came to the hospital for demonstration at a Clinical Meeting of the New South Wales Branch of the British Medical Association. His case excited considerable interest; discussion with physicians and surgeons as to his future prospects and the possible benefit of various surgical procedures confirmed me in my opinion that it was wise for him to content himself with his then quite satisfactory ability to earn his livelihood. He is obnoxious to reinfection of the partial pneumothorax through the evidently wide bronchial opening into it; but this danger seems less than that of the extensive operation which would be required to close down the chest wall to the contracted lung; again, even if the thick pleura could be successfully peeled off, I fear the lung would not, after ten years, expand again so as to fill the thorax.

Reviews.

PHYSICAL SIGNS IN CHEST DISEASE.

WE have before us the now well known treatise on physical diagnosis of the heart and lungs in health and disease, by Professor Norris and Professor Landis, of the University of Pennsylvania. It has now reached its fourth edition, the first having appeared twelve years ago. For a book of its size, over nine hundred pages, this shows that it fulfils a definite want. Unlike most medical books, its size is due in large measure to the illustrations, of which there are over five hundred, many occupying an entire page. For its illustrations and figures alone this book is worth a place in every physician's library, particularly of those who have the responsibility of teaching students.[1]

Although we are fain to consider "Norris and Landis" as an advanced book for the specialist, the general practitioner would obtain much to delight and instruct him by dipping occasionally into its pages.

Roughly, the first third of the book comprises the physical examination of the lungs and circulatory system, the remaining two-thirds being devoted to the diseases of these two systems.

Professor Norris deals with "The Examination of the Lungs" in twelve chapters. Correctly, he emphasizes the value of inspection, that method so much neglected by students. As we read, it soon becomes apparent how greatly do pictures relieve the tedium. The appeal to the visual sense is constant and invaluable as an aid to memory. For instance, a long description of dulness is followed by a photograph of a frozen section in which all shades of dulness are represented. We remember the picture and promptly forget the text. There is, as a matter of fact, tremendous detail of description, often more academic than practical. On the other hand, captions of physical signs pointing to portions of a photograph, give a false impression of simplicity of diagnosis. In auscultation the author makes bronchial synonymous with tubular breathing. A worse break with British teaching is his division of broncho-vesicular breathing, that refuge of the clinically timid, into physiological and pathological —hair splitting, surely!

Accompaniments also are divided in a way foreign to our teaching and the nomenclature they adopt is not to be recommended. The importance of the whispered voice sound is well emphasized, with the advantage over the spoken voice, that "an increase in intensity speaks more strongly in favour of actual change in the pulmonary tissue." This is a good point.

Radiography frankly he damns with faint praise. "The ordinary methods of diagnosis are far superior to the X ray in cases of incipient or early pulmonary tuberculosis. The X ray is rarely of much use as an early diagnostic method in cardiac or pulmonary diseases." Thus Professor Norris fairly flings down the gauntlet to the radiologist. But colleague Landis, some two hundred pages later, speaking of pulmonary tuberculosis, states: "The X ray becomes of the greatest importance in those cases in which the clinical procedures have failed definitely to establish the presence or absence of disease. It may cease to be an accessory agent and be the actual means of determining the cause of the trouble"; with which the radiologists may take consolation.

Professor Norris also contributes Part 2 on the examination of the circulatory system. Excellent *post mortem* sections are again illustrated. Curiously, inspection here receives scant mention. In the chapter on palpation the term "cardiac impulse" is employed, and not apex beat; it is regarded as being due to increased tension in the wall of the right ventricle, about one inch to the right of the anatomical apex. However, the cardiac impulse itself is regarded as of little importance clinically, dependent largely on individual variation. This chapter is rather poor.

In the chapter on cardiac arrhythmia Sir Thomas Lewis's work has been closely adhered to, though some new words appear of doubtful value, such as "allorhythmia" for a regular irregularity, and "parorhythmia." Electrocardiography is well dealt with by Dr. Krumbhaar and, as

[1] "Diseases of the Chest and the Principles of Physical Diagnosis," by G. W. Norris, A.B., M.D., and H. R. M. Landis, A.B., M.D., Sc.D.; with a chapter on the Transmission of Sounds through the Chest by C. M. Montgomery, M.D., and a chapter on the Electrocardiograph in Heart Disease, by E. B. Krumbhaar, Ph.D., M.D.; Fourth Edition, revised; 1929. Philadelphia: W. B. Saunders Company; Melbourne: James Little. Royal 8vo., pp. 954, with illustrations. Price: 50s. net.

befits its importance in modern medicine, considerable detail is given. An admirable summing up on its value to the clinician concludes "it is at best but one of several aids to the discriminating physician." Than which no more can be said.

Much detail is given on the need for accuracy and the significance of percussion. Is the shape of the heart dulness, however, really of much clinical importance? In the chapter on auscultation, ten pages are devoted to heart sounds and forty to heart murmurs. Shade of Mackenzie! The author offers an entirely new theory on the cause of the presystolic murmur in mitral stenosis which is academically interesting. The last three pages, on "practical considerations," are the best in this part.

To Professor Landis falls the largest section, occupying nearly half the book, that on respiratory diseases. Practical points such as those on chronic bronchitis enhance the value of his writing. "As a primary affection, chronic bronchitis is encountered much less frequently than is usually thought." And again, "until disease of the heart, kidney or other pulmonary lesion are excluded a diagnosis of chronic bronchitis alone is untenable."

He correctly criticizes the teaching that bronchiectasis is an uncommon disease, modern technique, including the use of "Lipiodol," having revealed its frequency. There is a very good section on foreign bodies in the air passages, based largely, of course, on the work of Chevalier Jackson.

One hundred pages are devoted to tuberculosis. Illustrations and diagrams as usual are plentiful, and the author maintains a well balanced opinion. An exception is the following dogmatic statement with which we are in definite disagreement. "The mathematical chances of tuberculosis being present are overwhelming in the case of any individual who has an hæmoptysis." In our experience there are many other "chances," bronchiectasis being not at all unlikely. We are offered an excellent appraisal of the value of physical signs, due stress being laid on the importance of inspection. An interesting observation he makes is the bad prognostic import of violet coloured nails. Due warning is given of the danger of relying on auscultation alone. This merits emphasis and frequent repetition. Much is made of physical signs in cavity formation, though post-tussive suction is not mentioned. In this disease alone are we given some notes on prognosis, as though the author could not resist the temptation. He was ill-advised, as they are very skimped and incomplete. On the other hand, here alone is differential diagnosis not given—a serious omission.

The two pages on X rays suggest that the average radiologist in the United States of America has a standard below that in Australia, though we agree on the importance of accurate technique and wide experience, without which radiology of the chest is a menace rather than a help.

In regard to tracheo-bronchial tuberculosis in children, Dr. Landis looks on symptoms as "worthless" and examination for signs as "a hopeless proceeding." This is a little surprising, but distinctly refreshing. The tuberculin test and X ray examination he regards as essential for diagnosis, which is in accord with most modern views.

Eighteen pages are taken up with mycotic infections of the lungs and bronchi, perhaps more space than they merit.

Hydatid disease is very poorly treated. The bias of both authors against X rays shows out often, which may explain, though it does not condone the following gross blunder and dangerous advice in discussing diagnosis: "Exploratory puncture offers more certain proof than the X rays." Such a statement at examination by any Australian student would prove a bar to graduation. It is surprising that no mention is made of eosinophilia or any of the recently perfected allergic and serological tests.

Syphilis of the respiratory tract offers a section on laryngeal involvement, though no mention was made earlier of the far more important tuberculous disease of the larynx. Syphilis of the lung is now a recognized clinical and pathological entity; the author gives one of the best descriptions we have read of this disease.

The chapter on hæmothorax illustrates a difficulty the authors have in such a book as this. We find Dr. Landis here trembling on the verge of treatment and eventually toppling over in offering advice on thoracotomy or aspiration. "It is usually advisable on the third or fourth day to remove as much of the outpoured blood as possible." This is good advice, but it raises a doubt as to the wisdom of divorcing the important subject of treatment from the remainder of the practice and principles of medicine.

The last chapter is somewhat of an innovation, dealing with diseases of the diaphragm. The anatomy and physiology are discussed, though the latter would appear to be given undue importance in view of the absence of ill effects following the modern operation of phrenic avulsion; bilateral avulsion has been performed without inconvenience to the patient. Sections then follow dealing with functional disturbance of the diaphragm, hernia, evisceration, eventration, subphrenic abscess and diaphragmitis. Those who think the latter a fanciful term, should read this, when they will be convinced of its importance as a complication of pulmonary disease.

The authors combine in the last part on circulatory disease. The section on pericarditis is notable for the excellent photographs and for the clear exposition of the difficulty in diagnosing all forms of this malady.

In the chapter on myocardial disease, there is an excellent introduction. The correct balance is soon struck between valve defects and muscle efficiency. "Although the term 'heart disease' is, in the minds of many, associated with a valvular defect, it cannot be too strongly emphasized that the ability of the heart to carry on its functions is dependent almost entirely on the condition of the cardiac muscle." This sentence might have been written by Mackenzie. We heartily endorse the authors' statement that: "The term myocarditis is not altogether a satisfactory one." But alas, their courage failed and so: "It is retained because, by common usage, it implies disease of the myocardium." Undoubtedly they should have separated the degenerative processes from the inflammatory lesions. From the point of view of pathology it is quite incorrect, for instance, to state that coronary thrombosis leads to a "localized acute myocarditis." In discussing "goître heart" they fail to distinguish the early and often temporary toxic myocarditis from the later permanent myocardial degeneration, an important prognostic point.

Notwithstanding their remarks, the chapter on endocarditis occupies twice as many pages as that on myocarditis. In discussing the streptococcus theory of rheumatic fever, reference is made to the work on this by J. C. Small in America, published in 1927. It is strange indeed to find no mention of the work of Poynton and Paine, which antedates this by many years.

Due emphasis is laid on the rarity of aortic stenosis and on the diagnostic importance in this lesion of a feeble aortic second sound. In congenital heart disease the heading "imperforate ventricular septum" is an obvious mistake.

In *angina pectoris* the traditional subdivision is followed into true and false angina. To this latter paradoxical term Mackenzie's "secondary angina" is much to be preferred. Diseases of the aorta are fully dealt with under the subtitles of acute aortitis, syphilitic aortitis and chronic aortitis. Under the last named heading we find, however, a discussion on arteriosclerosis in general. There has been a confusion of the part with the whole and arteriosclerosis should have been the heading.

The chapter on aneurysm of the aorta occupies no less than thirty pages. Its importance in this book is to be attributed largely to the prevalence of this disease in the negro population; four out of the six photographs of patients are of negroes.

In these days of increasing reliance on accessory aids to diagnosis the authors have put up a magnificent defence, a glorification, as it were, of physical signs. Laennec and Auenbrugger can have had no worthier disciples, in the United States at least. If the day ever comes when the stethoscope and the percussing finger, like the hand plough and the hansom cab, are relegated to the limbo of the past, future clinicians will see "under glass" the laborious methods of their forefathers to establish an ofttimes elusive diagnosis with their unaided senses, enshrined in the work of Norris and Landis.

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CLINICAL PATHOLOGISTS.

IF a little consideration be given to medical practice as a whole, it will be recognized at once that there is a peculiar inequality in regard not only to the incomes earned by practitioners devoting their attention to different branches of medical practice, but also to the place accorded to practitioners of each type in the public esteem. Surgeons, both general and special, are able to command large fees; they take, as it were, the life of the patient into their hands and by direct attack with the scalpel can make or mar his future health. The public recognizes the dexterity of the surgeon rather than the maturity of judgement directing his actions and is content to place him on a pedestal and pay the fees required by him. The physician, possessed necessarily of a type of mind different from that of the surgeon, does not cut the disease out of his patient, but helps the patient to overcome it himself. His problems may be more obscure than the surgeon's and his responsibility in giving advice is often much greater—the surgeon can always "explore the abdomen and have a look." The physician seldom if ever gains a competence comparable to that of the surgeon, nor is he looked upon by the laity as being quite so brilliant, so necessary or so worthy of recompense. His financial position, however, is generally regarded as satisfactory. As for the general practitioner who bears "the burden and heat of the day," he undoubtedly is relegated to the third place; but it must be remembered that if the physicians are, as Dr. C. H. E. Lawes suggested, the artillery, pouring "a continuous fire of pills and potions with bombs of serums and vaccines at the advancing foe," and if the surgeons are the cavalry of the medical army, the general practitioners make up the infantry on whom victory ultimately depends. Like the Australian privates during the Great War, Australian general practitioners, compared with those of other countries, earn a relatively good income. The arm of the service most frequently overlooked is the intelligence department—that of the clinical pathologist. The term pathology is used here to denote not only morbid anatomy, but also bacteriology, immunology and biochemistry in their clinical applications. An army without an intelligence department is quite ineffective and the activities of the enemy are so varied and widespread that more and more basal information is becoming necessary to the fighters of disease.

The clinical pathologist does not receive adequate recognition at the hands of either the public or his fellow practitioners. This is reflected in the fact that at any rate in one Australian university professors of bacteriology and of pathology are not paid so highly as professors of medicine or surgery. The general public regards the clinical pathologist as a man who mixes things in a test tube or looks at germs under a microscope and charges a fee for describing what he sees. It matters little whether the clinical pathologist is put by the laity on a pedestal of wonder. It is not necessary to the progress of medical science that any of its practitioners should be unduly regarded—a proper sense of proportion would lead to fewer personal disappointments. It does matter, however, that the importance of clinical pathology should be recognized and that the pathologist should receive due consideration at the hands of other practitioners.

Throughout Australia the examination of pathological specimens, the preparation of vaccines, the culture of microorganisms, chemical examination of

the blood and other body fluids, and other investigations are carried out for private medical practitioners by both government departments and laboratory workers in private practice. The government departments exist so that investigations required by public hospitals and other public institutions may be carried out. In certain circumstances examinations are carried out at government laboratories for private practitioners, but it is understood that this is done only when the patient concerned is not in a position to pay a private medical practitioner to do the work or when no medical practitioner capable of doing it is available. The number of investigations carried out at any one of these departmental laboratories in one year is enormous, as may be seen on perusal of their annual reports. For example, it is stated in the last report of the Director-General of Public Health of New South Wales that in 1928 42,677 examinations were made at the Bureau of Microbiology, Sydney. Of these 23,323 were made for private medical practitioners, and of the total number of examinations 9,072 were Wassermann tests and 8,380 Kahn flocculation tests. It has been obvious for quite a long time that medical practitioners are not carrying out their part of the bargain. Specimens are frequently submitted on behalf of patients who could well afford to pay to a medical practitioner either the full fee or a modified or "intermediate" fee. This is not right. Medical practitioners are loud in their protests at any encroachment by governmental departments or other bodies on what they regard as their own particular preserves, they object to hospital abuse and they stand in perpetual fear of bogies such as nationalization, and yet some of them are prepared to take work that rightly belongs to the consulting pathologist and give it to a government department. No practitioner should submit specimens to a departmental laboratory on behalf of a patient from whom he is receiving full private fees; neither should he offer an "intermediate" fee to a practising pathologist if he himself is in receipt of full fees. Unfortunately there are in every community unworthy individuals who exploit others to their own advantage; medical practitioners need not add to their number.

Current Comment.

GEOGRAPHICAL DISTRIBUTION OF RHEUMATIC FEVER.

FOR generations past authors of text books on medicine have consistently informed readers that rheumatic fever is ubiquitous. From time to time various observers have expressed doubt concerning the occurrence of the disease at all in tropical countries or in some portion of the tropical zone in particular. As part of the transactions of the seventh congress of the Far Eastern Association of Tropical Medicine in 1927, U. P. Basu presented a paper in which he mentioned the rarity of rheumatic disease of the heart in Bengal. In the course of the discussion which followed, Stott quoted Rogers as having proven the absence of rheumatic fever from Calcutta.

The incidence of rheumatic fever is affected by a definite seasonal influence. In London, for example, it is said to be most common in a cold, wet autumn after a hot, dry summer, though, as noted in a special abstract in this journal on May 7, 1927, the results of an investigation undertaken on behalf of the Medical Research Council indicated that in Great Britain it most commonly occurred during the winter. In New York it is said to occur with greatest frequency in the spring time after a severe winter. But in the tropics there are no seasons comparable to the seasons of the temperate zones. Save in some few more blessed areas, there is no cold weather, and the only features marking a difference in seasons may be an alteration in rainfall and humidity and in the direction of the prevailing wind. Some parts of the tropics are free of many of the acute infectious diseases, and some pathological conditions are peculiar to warm countries. The suggestion that the whole of the tropics may be free of rheumatic fever is therefore not so fantastic as it may appear at first glance. At any rate, it may be taken quite definitely that the disease is rare in the tropical zone. There are so many conditions which may simulate rheumatic fever that the likelihood of incorrect diagnosis or confusion in terminology as a source of statistical error cannot be overlooked, and possibly careful and searching analysis would result in a reduction of the reported incidence perhaps to nil.

The view that rheumatic fever does not occur at all in the tropics is championed by J. Tertius Clarke.[1] He points out that the term "tropics" is loosely applied often to countries which are partially or wholly without the tropical zone; for example, Egypt, China and India are usually said to be tropical, though with accuracy only a lesser portion of each could be so designated. Clarke's definition of the tropics as an area lying between the lines of Cancer and Capricorn is geographically accurate, but for the purpose of consideration of the relation between climate and disease incidence it is faulty, as a study of the isotherms will show.

[1] *Journal of Tropical Medicine and Hygiene*, September, 1930.

In 1899 Cantlie remarked that he had never seen rheumatic fever in any hospital in China, India or Egypt, and he further declared that he had never seen a rheumatic cardiac lesion *post mortem* in a native of a warm country. This despite that the incidence has been said to be very high in Egypt. Writing of India in 1928, Leonard Rogers remarked on the absence of rheumatic endocarditis and the rarity of disease of the mitral valve. The evidence of two such authorities as Cantlie and Rogers cannot be lightly regarded. Again in 1928 Calvert and Sutherland, while admitting that rheumatic fever is rare in Calcutta, nevertheless declared that they had seen chorea, endocarditis and rheumatic fever in young Bengali children. Clarke points out, however, that the majority of the inhabitants of Calcutta, which is only about one degree south of the tropic of Cancer, come from outside its boundaries and that the finding in the city's hospitals of a very few patients suffering from rheumatic fever cannot be regarded as a serious argument against the view that the disease does not occur in the tropics.

During his thirty-three years' experience in the tropics Clarke made observations on 150,000 hospital in-patients. Though he consistently sought evidence of rheumatic fever he found none either clinically or at *post mortem* examination. Duncan, who conducted six hundred *post mortem* examinations in Singapore, and Fletcher, who had many years' experience as pathologist in Kuala Lumpur, support his views. In the report for the year 1925 the incidence of rheumatic fever among the total number of patients treated in Panama was given as 0·11%, but Clarke points out that the population of this region is cosmopolitan and can contain but few people of twenty years of age who were born in the country. Further, the figure reported is so low that it might be accounted for by occasional inaccuracies in diagnosis.

Inflammation of the joints is a common occurrence in the tropics as elsewhere, and swelling and pain of the joints are frequent accompaniments of many diseases. Typhoid fever, Malta fever, relapsing fever, cerebro-spinal fever, trichinosis, subacute osteitis *et cetera*, all may simulate rheumatic fever. The anæmia of ankylostomiasis may be accompanied by swollen joints and heart murmurs, and if complicated by malaria would present a clinical picture which might readily be mistaken for rheumatic fever. Hæmic murmurs are, of course, common in the tropics. Clarke thinks that they may account for the report of 102 cases of mitral disease and none of rheumatic fever during a period of twelve months in Nigeria. He points out, on the other hand, that in Jamaica and British Guiana rheumatic fever is reported to be rather common, though no mitral disease is reported from either country. He argues that there must be some error here, as the most common cause of disease of the mitral valves is rheumatic fever, and rheumatic fever is followed or accompanied by mitral disease, perhaps invariably.

Clarke points out that if the disease does occur in the tropics, its existence may readily be proved *post mortem* by finding hearts affected with rheumatic endocarditis, of which the chief pathological feature is the Aschoff body. Finally, he remarks that even if the statistics quoted are correct, the difference between the incidence in temperate or cold climates and in the tropics is very great and indicates the existence of some factor, common in one climate and rare in another, for which search should be made.

Clarke has made a very thorough study of his subject with interesting and perhaps valuable results. The possible absence of rheumatic fever from the tropics is not merely of interest to the student of tropical hygiene. If it be proven, a way may thus be opened up for the acquisition of much knowledge concerning the ætiology of the disease. The problem merits the study of medical practitioners in tropical countries, but it should be approached on scientific lines. Clarke's definition of the "tropics" does not appear to be rational for the purpose. It is difficult to conceive how two imaginary lines encircling the globe may act as a barrier to the spread of disease, but it may be readily believed that the boundary area of certain climatic conditions could so act. It is possible that the area of distribution of rheumatic fever may not be bounded by any definite isotherms, but may be found to follow the distribution of some other factor, just as dengue fever coincides with the distribution of the mosquito *Aedes argenteus*.

EMPHYSEMA FOLLOWING TONSILLECTOMY.

AMONG the more unusual complications of tonsillectomy is emphysema. It is therefore interesting to note that F. H. von Hofe reports the occurrence of three cases.[1] They occurred in association with removal of the tonsil by the La Force-Sluder method and are important on account of the different causes assigned to them. The patients were aged two, four, and two and a half years. One child cried and strained a great deal and it is thought that the air may have entered the tissues following perforation of lung vesicles. Another child was very quiet while the anæsthetic was given and it is held that the air may have entered after perforation of the tonsillar fossa bed. In the third instance the air is thought to have entered the tissues by diffusion after being forced into Wharton's duct. The disturbance was on the right side in each instance and recovery was uneventful. As far as we can discover, no instance has been reported in this journal. von Hofe makes no suggestion as to how the complication may be avoided. Sometimes, of course, emphysema might occur in spite of all precautions, but placid anæsthesia and neat, clean operating will generally obviate unusual happenings.

[1] *The Journal of the American Medical Association*, September 27, 1930.

Abstracts from Current Medical Literature.

PHYSIOLOGY.

Caffein Diuresis.

EXPERIMENTS by E. B. Verney and F. R. Winton (*Journal of Physiology*, April, 1930) on isolated kidneys of dogs perfused by means of the heart-lung preparation suggest that the diuresis produced by caffein is essentially a vascular phenomenon. The changes in the chloride and urea content of the urine, when diuresis is caused by this drug, are the same in kind and magnitude as when a similar increase in the rate of urine flow is produced by a rise in perfusion pressure. An increase in blood flow through the kidney is an integral part of the action of caffein and may be marked, even when little or no diuretic effect is present. The authors point out that these results can be explained by supposing that the drug induces dilatation of both *vasa afferentia* and *efferentia* of the renal glomeruli, but that in certain circumstances the effect on the former is the greater and as a consequence the glomerular pressure is increased.

The Effect of Pituitrin on the Excretion of Water through the Kidney.

EARLIER investigations have shown that pituitrin influences the renal secretion without altering the circulation through the kidney. The diuresis which normally follows the ingestion of considerable quantities of water or saline solution, is inhibited for several hours by the simultaneous administration of pituitrin. L. T. Poulsson (*Zeitschrift für die gesamte experimentelle Medizin*, May, 1930) has analysed as far as possible the changes in the blood and in the concentration of several urinary constituents accompanying this antidiuretic effect. Most of the experiments were on human subjects. In the fasting subject in whom no recent absorption of water has occurred, pituitrin most characteristically causes a concentration of the blood as determined by its hæmoglobin content. The change is, however, rarely greater than 5%. No appreciable alteration in the rate of urine flow occurs. Associated with the inhibition of diuresis by pituitrin after the ingestion of water there occurs a small degree of hydræmia. By determining the amount of the blood and urinary creatinine and the rate of urine flow Poulsson estimates the rate of filtration in the renal glomeruli and finds that it is uninfluenced by pituitrin and that the antidiuretic effect is due to increased water absorption in the tubules. After pituitrin injection the absolute quantity of urea excreted as well as its concentration in the urine decreases. From an analysis of the experimental results the author concludes that pituitrin facilitates absorption of water in the proximal tubules. He also throws doubt upon the generally accepted idea that an hydræmia following the ingestion of water causes diuresis because of a diminution of the osmotic pressure of the plasma proteins and a consequent increase in the effective filtration pressure in the glomeruli.

Experimental Diabetes Insipidus.

WHETHER lesions of the pituitary gland or of the neighbouring hypothalamus are primarily responsible for the occurrence of *diabetes insipidus* is still a debated question. C. P. Richter (*Brain*, April, 1930) has described a series of experiments in which puncture of the hypothalamic region in rats resulted in the production of polyuria and polydipsia. A most convenient surgical approach to the pituitary and hypothalamic regions is described in some detail. Deep dissection between the masseter and digastric muscles exposes the auditory bulla and by stripping the periosteum medially from this structure the basisphenoid is exposed. Removal of the bone immediately in front of the spheno-occipital suture exposes the pituitary gland, while puncture some three millimetres anterior to the suture injures the hypothalamus without interference with the pituitary. This approach is a distinct improvement on the transbuccal route and should be available in a number of different experimental animals. The production of hypothalamic lesions immediately anterior to the pituitary gland caused a permanent increase in the water intake; in one instance the animal consumed almost twice its body weight of water daily. This great increase of fluid intake was accompanied by a corresponding increase in the urinary output. An analysis of the variations in the degree of the polyuria and polydipsia indicated that changes in the water consumption preceded similar fluctuations in the urinary secretion. No changes in the sexual cycle of the animals were observed and the author considers this to be evidence that no damage to the pituitary gland resulted from the operative procedure. Histological evidence of the extent of the lesion in the brain stem is at present lacking, but is to be given in a later paper.

The Measurement of Red Cell Volume.

THE most commonly used method for the determination of red cell volume is that of centrifuging and then measuring the relative volumes of the separated mass of cells and of the supernatant plasma. E. Ponder and G. Saslow (*Journal of Physiology*, Volume LXX, Number 1) have indicated the extensive errors to which this method is liable. The final reading is dependent upon both the speed and the time of centrifuging and even the standardization of the end point by the application of Koeppe's criterion, the appearance of transparency in the column of cells as if laking had occurred, does not give consistent results. Errors as great as 20% may occur. Nevertheless the method is of use in the determination of relative change in cell volume if all observations are made with a constant technique. The fallacies of the refractometric method are principally the assumption that the refractive index of a mixture is the mean of the indices of its constituents and the fact that dilution with different "isotonic" fluids leads to markedly different values for the cell volume in the same sample of blood. Even if the more accurate formula for the relation between refractive indices of a mixture and its constituents is employed gross errors arise from the inaccuracies of measurement of the density of the various fluids used. Even the theoretical application of the formula, which is derived for solutions of crystalloids, to the colloidal system of the plasma is not justified. Methods entailing dilution of the blood with saline solution containing glucose or some dye are based on the quite unwarranted assumption that the added substance does not penetrate the corpuscle wall. Viscosity methods require dilution of the blood with isotonic fluids, but there is abundant evidence that such fluids may cause marked changes in the volume of the red cells. The authors describe in detail the application of a dilution method introduced by Stewart in 1899. In this the blood is diluted with some of its own plasma after having been hæmolysed by freezing and thawing. The stroma of the laked corpuscles is removed by centrifuging and the clear solution of hæmoglobin obtained is used as a diluting fluid for a measured volume of blood. The hæmoglobin in the diluting fluid and in the plasma of the diluted blood is estimated as acid hæmatin. This method, unlike others, gives consistent results for the corpuscle volume of a given sample of blood and the maximum error in a single determination is $\pm 3\%$.

Physiological Effects of Sympathetic Ganglionectomy.

THOMAS LEWIS AND E. M. LANDIS (*Heart*, Volume XV, Number 2) describe the changes in the skin and local vascular reactions of the human subject after partial excision of the cervical sympathetic chain. As judged by the failure of local faradization to produce goose skin, it appears that degeneration of the post-ganglionic fibres is complete from eight to seventeen days after removal of the corresponding ganglia. Pilocarpine produces sweating in the area where post-ganglionic degeneration has occurred. It is suggested that when this drug fails to produce a response in the denervated area while sweating occurs in the normal zones the stimulus to secretion has been reflex in nature. After sympathectomy the denervated area of skin becomes paler and warmer. There appears to be a simultaneous increase in the tone of the small arteries. Local cooling produces a more marked fall of surface temperature on the normal than on

the denervated side. If generalized flushing is produced by overheating, the temperature of the areas deprived of sympathetic innervation remains almost unchanged. Emotional blushing is absent from the denervated areas. The subcutaneous injection of histamine results, as is well known, in a generalized flushing of the skin. This is more pronounced on the side on which the sympathetic trunk has been removed than over the corresponding areas on the normal side. Consequently the temperature difference previously existing between these areas becomes more pronounced. From observations on the cutaneous veins it is concluded that these vessels are normally under a tonic sympathetic control. A dilatation of the cutaneous veins occurs, however, in response to heat, even in the denervated areas. After degeneration of the sympathetic nerves the transient vasodilatation which normally follows light pressure upon or gentle stroking of the skin still occurs. Spasm of the digital vessels may still be produced in Raynaud's disease by local cooling after extirpation of the sympathetic trunk responsible for the innervation of the affected part. This indicates that the primary cause of spasm does not lie in abnormal vasomotor impulses reaching the vessels through the sympathetic nerves.

BIOLOGICAL CHEMISTRY.

Phlorizin Diabetes.

EPHRAIM SHORR, ROBERT O. LOEBEL AND HENRY B. RICHARDSON (*Journal of Biological Chemistry*, April, 1930) have undertaken an investigation on the nature of phlorizin diabetes. Previous work is outlined in the introduction. The accuracy of the respiratory quotients obtained with the Barcroft-Warburg micro-respirato r y apparatus was first determined and was found to be ± 0·014. Excised renal tissue from the rat was used. All the quotients fell between 0·705 and 1·00, implying that excised tissues utilized foodstuffs in the same proportions as does the intact animal, though the excised tissue is deprived of nerve connexions and circulation. During the experiments no evidence was obtained that one kind of foodstuff is converted into another. The excised renal tissue of the normal rat was found to respond to the presence of glucose by an increase in oxygen consumption and a corresponding rise in the respiratory quotient. Excised renal and testicular tissue of rats treated by phlorizin, with and without phlorizin in the medium, responded to the presence of glucose, in the same way as normal tissue, by increase in oxygen consumption and corresponding rises in the respiratory quotient. The evidence which leads these workers to state that these changes are due to the oxidation of glucose is discussed in the original paper. Excised renal tissue of normal rats in the presence of varying concentrations of phlorizin suffered no depression of the respiratory quotient. The average of the quotients, 0·850, indicated that carbohydrate was furnishing about 50% of the energy. The authors conclude that the results of these experiments are against the theory that phlorizin interferes directly with the oxidation of carbohydrate by the cells in general.

Fat Metabolism.

EDITH H. MACARTHUR (*Journal of Biological Chemistry*, June, 1930) has used the method of Gage and Fish to gain further evidence on the rate of digestion of fats. In this method, by means of the dark field microscope, the visible lipæmia of the blood, due to small fat particles called chylomicrons, can be studied during the digestion of a fat by the use of one drop of blood each hour. The subjects used in this investigation were college women in good physical condition and healthy white rats. The subjects were kept without food for twelve to fifteen hours preceding the experimental period. The rats to be tested were generally fed with carbohydrate food. A drop of blood was taken every hour during the digestive cycle for seven or eight hours and was examined with the dark field microscope. No difference was found in the chylomicron count of blood taken from the central and peripheral circulations of the rat; consequently blood was taken for these curves from the peripheral circulation. The digestion curves obtained on the chylomicron counts are critically considered. Under the conditions of the experiment the author found that butter from cow's milk and cod liver oil are taken up by the blood much faster than are the same amounts of either butter from goat's milk or butter from cow's milk when eaten with bran.

Nitrogenous Substances in Zinc Filtrates of Human Blood.

MICHAEL SOMOGYL (*Journal of Biological Chemistry*, June, 1930) reports that protein-free blood filtrates prepared by the Hagedorn Jensen technique contain smaller quantities of reducing non-sugars than tungstate filtrates and that a new method of deproteinization by zinc yields filtrates practically free of such substances. He also suggests that glutathione is responsible for a substantial fraction of the residual reduction in tungstic acid filtrates and at the same time demonstrates that thioneine, another constituent of tungstate filtrates, also reduces the Shaffer Hartmann reagent. He infers from the correlation of the two observations that lower quantities of reducing non-sugars would go hand in hand with lower nitrogen contents in the several sorts of blood filtrates. He has shown in this present work that blood filtrates prepared by deproteinization with zinc contain substantially smaller quantities of nitrogen than tungstate filtrates. He found that non-protein nitrogen in zinc filtrates varies normally between 11 and 21 milligrammes per 100 cubic centimetres of human blood, the average being 17%. In cases of nitrogen retention the non-protein nitrogen was in excess of 21%. Uric acid was found to be entirely absent in zinc filtrates, while creatinine was slightly less and urea showed identical values in zinc and tungstate filtrates. The author therefore concludes from these results that zinc filtrates prepared for the determination of the true blood sugar content are also adequate for the determination of non-protein nitrogen urea and creatinine content.

Blood Sugar Values.

MAURICE J. DIONNE AND JACOB J. ARENSTAM (*Journal of Biological Chemistry*, June, 1930) have investigated the concentration of sugar (total reducing substances) in the capillary blood of normal young women both for fasting individuals and for a group engaged in their normal activities over a period of twenty-four hours. The sugar concentrations were measured by the Folin ferricyanide method. Each individual subject maintained a very constant concentration level while in bed and asleep. The average level found for the entire group was 94 milligrammes *per centum* and the majority of the individuals had values close to this. The data collected by these workers with young women as subjects are in close agreement with those obtained by Trimble and Maddock for young men and they consider that they are justified in stating that when the Folin micro procedure is used for measuring the sugar content of the whole blood and the subject is in a basal condition, the normal fasting values should lie within or very close to the range 90 to 100 milligrammes per 100 cubic centimetres and that the basal range for blood sugar values probably extends over a smaller area than has been generally accepted.

Basal Metabolism.

JESSIE TILT (*Journal of Biological Chemistry*, April, 1930) has studied the basal metabolism of fifty-two subjects at the Florida State College for Women. The subjects were between the ages of seventeen and twenty-five years; the majority were students who had studied energy metabolism. They showed a fine spirit of cooperation and at no time was there evidence of any psychic disturbances which might influence the results. The metabolism determination was not made during the menstrual period. The average metabolism of the group was −9·9% (Harris-Benedict standards) and −10·6% (Aub Du Bois standards). Eight of the subjects were observed for a period extending over three to six months and from these few data it appears that there is no change in metabolism with change in season. The figures obtained by this worker closely resemble the figures obtained by Hindmarsh in Australia. Eight of the subjects who indulged in greater physical exertion, had an average metabolism agreeing closely with the average of the entire group.

Special Articles on Diagnosis.

(Contributed by Request.)

XIX.

NASAL SINUSITIS.

In considering the diagnosis of nasal sinusitis on general lines, we need to differentiate between the acute condition and the chronic.

Acute Nasal Sinusitis.

The patient shows the general signs of an acute infection. A severe cold in the head is present. Locally the nose is obstructed for breathing. There is a thin serous discharge from the nose, causing excoriation of the *alæ nasi*. The mucous membrane of the nose is swollen and engorged and the mucosa lining the sinuses is also inflamed. The ventilation and drainage of these sinuses are blocked, and thus headaches, usually more marked over the frontal region, are caused. Tenderness on percussion over the region of the affected sinuses is present.

As this heavy cold runs its course, the nasal discharge thickens and becomes purulent and later muco-purulent. Usually the associated sinusitis subsides as the acute nasal infection subsides. But sometimes one or more of the sinuses remains in an acutely suppurating condition. This is shown to be present by the persistence of the purulent nasal discharge from the affected side. Cacosmia may be present. Headache and the tenderness over the affected sinus persist. The pain caused by an acute sinus infection tends to appear at regular intervals, frequently being most marked towards the end of the morning and diminishing towards evening. It corresponds in severity to the increase of pressure caused by the pus inside the sinus, becoming worse up to the point when this pus forces its way through the ostium into the nose and then gradually lessening in severity. On account of the drainage of the purulent discharge posteriorly there are soreness of the throat, huskiness of the voice, and cough which is often most marked on lying down. The temperature is frequently raised and there are general signs of toxic absorption.

If we examine such a patient, we find the following:

The Oropharynx: The tonsils are hyperæmic and rather swollen. The mucosa on the posterior wall is congested and may show a slightly glazed appearance. Streaks of pus are often present. The lymphoid tissue of the lateral pharyngeal bands are swollen and inflamed, particularly on that side which corresponds to the side of the nose that is suppurating.

Post-Nasal Space: Posterior rhinoscopy shows the mucosa of the post-nasal space to be red and engorged and pus and muco-pus is present.

Larynx: Examination of the larynx generally reveals a congested mucosa and often pus lying about the glottis.

Nose: On anterior rhinoscopy the affected side of the nose shows swollen, enlarged turbinates and yellow pus is present. In order to obtain a better inspection the nasal passages may be sprayed with a 2% solution of cocaine with or without a few drops of adrenalin chloride solution (one in 1,000). This will enable the examiner to get a better view of the middle turbinate region.

Pus coming from the middle meatus from below the middle turbinate indicates suppuration of one of the anterior sinuses, that is, frontal sinus, anterior ethmoid cells or maxillary antrum.

Pus coming from the maxillary antrum is found draining from below the middle turbinate over the inferior turbinate to the floor of the nose. On account of the swelling of the mucosa of the naso-frontal duct, ventilation and drainage of the frontal sinus on the same side are obstructed. Pain and tenderness over this sinus are very marked, usually more than over the acutely suppurating maxillary antrum. This often suggests that the patient is suffering from an acute frontal sinusitis, when the real predominating lesion is in the maxillary antrum, and it is special treatment of this sinus that relieves the frontal symptoms.

The frontal sinuses and ethmoids are less frequently left as an acute sinusitis at the end of a heavy cold on account of the more efficient ventilation and drainage of these cavities, due to their better anatomical position.

When the frontal sinus is suppurating, the pus is found higher up and more anteriorly in the middle meatus.

Should a posterior sinus, that is, posterior ethmoid cells or sphenoidal sinus, be affected, the pus will be found posteriorly between the middle turbinate and the septum, for the drainage comes *viâ* the spheno-ethmoid recess and the superior meatus.

The sphenoidal sinus, being like the antrum in regard to having its ostium situated in a position unfavourable for efficient drainage and free ventilation, is prone to retain an acute sinusitis. The symptoms generally noted are severe occipital headache, and pain and tenderness down the back of the neck. Blurring of the vision from involvement of the optic nerve is not uncommon.

Posterior Rhinoscopy: In the case of an anterior sinus suppuration, that is, maxillary antrum, frontal sinus or anterior ethmoid cells, posterior rhinoscopy will show pus to be present over the posterior end of the inferior turbinate on the superior surface of the soft palate and tracking down behind the posterior pillar of the fauces.

In the case of a posterior sinus (sphenoidal sinus and posterior ethmoid cells) suppuration, the pus will be seen entering the post-nasal space through the superior arch of the posterior choana above the level of the posterior ends of the middle turbinates. It tends to lodge in the roof of the naso-pharynx and to run down the posterior wall.

Having determined the site of the purulent discharge, we next use the transillumination test.

The Transillumination Test.

The Maxillary Antra: The patient's head must be in complete darkness. The light is put into the mouth after any artificial dentures have been removed, and is kept strictly in the mid-line. The lack of translucency, especially towards the outer half of the infraorbital margin, denotes an infected antrum. Should the other side not be affected, the comparison between the two sides makes the test more decided. The test may also be carried out by applying the light to the infraorbital region over the anterior surface of the antrum and noting the amount of translucency by looking into the patient's mouth.

The Frontal Sinus: In examining the frontal sinus, the light is pressed up against the tissues over the inner part of the floor of the sinus, that is, just above the internal canthus of the eye. The examiner's hand is placed on the supraorbital margins so as to shade the forehead above. In the case of acute frontal sinusitis the area of the forehead related to the sinus remains dark, whilst the unaffected sinus shows up as a reddish glow on the forehead. The septal partitions marking out different compartments stand out dark on the red background.

X Ray Films.

X ray films will show the presence of pathological swelling of the sinus mucosa, and pus in the cavity casts a distinct shadow with a fluid level.

Proof Puncture.

In the case of a suppurating maxillary antrum the diagnosis will be finalized by the presence of pus in the return lotion of a proof puncture and lavage. This minor operation is generally performed under a local anæsthetic, the patient being either in bed, sitting up, or in a consultation chair.

The following technique may be recommended. A straight wire wool carrier is taken and a piece of wool firmly wound on. This is dipped into a solution of one in 1,000 adrenalin chloride and saturated. Then it is dipped into some powdered (not crystallized) cocaine hydrochloride, about one half of one grain is usually enough. This forms a paste on the wool. If excess of fluid is present it is squeezed off, for one needs to prevent any of this concentrated solution from running back into the naso-pharynx and being swallowed, on account of the possibility of toxic symptoms developing.

The applicator is passed up under the lower edge of the inferior turbinate as high as its attachment to the lateral nasal wall and about 1·25 centimetres (half an inch) from its anterior end, the wool with its adrenalin paste thus being applied to the nasal surface of the naso-antral wall of the inferior meatus. This remains in place for about ten minutes, when anæsthesia is complete. A straight Lichtwitz trocar and cannula are then put into the nose in the same position as the applicator occupied and thrust through the naso-antral wall into the antrum. The Higginson's syringe is filled with "Glycothymoline" solution and connected to the cannula, and a continuous stream of lotion is syringed through the antrum from the bowl containing the lotion. The return flow from the nose is received into a kidney dish.

When pus ceases to come from the antrum and the wash from the nose is clear, the bowl of lotion is taken away. The lotion remaining in the syringe is forced through the antrum and a final few squeezes of the bulb drive some air through the antrum into the nose; it cannot, of course, empty the antrum of remaining lotion. The Higginson's syringe is then disconnected from the cannula and the latter withdrawn. The patient blows through the nose to clear it. An acute maxillary antritis following dental infection is diagnosed from the history, together with the signs and symptoms previously discussed in that of a case following an acute nasal infection.

Chronic Nasal Sinusitis.

Chronic nasal sinusitis may be divided into the suppurative type and the non-suppurative type.

Chronic Suppurative Nasal Sinusitis.

In chronic suppurative nasal sinusitis there are signs and symptoms associated with the more or less constant anterior and posterior nasal discharge, which is generally found to be present on inspection of the nose.

Nasal breathing becomes obstructed on account of the hyperplastic changes of the nasal mucosa. The hyperplasia is most marked in that region of the nose where the secretion from the infected sinus enters and drains away.

In anterior sinus suppuration the mucosal hypertrophy is found to be most marked in the region of the middle meatus. The anterior end of the middle turbinate is usually enlarged. The inferior turbinate also generally shows some hypertrophy. Nasal polypi are often present, springing from under the middle turbinate. These nasal polypi increase in size and number as the sinus suppuration continues. Posteriorly they tend to fill up the posterior choana and may be seen in the post-nasal mirror. Anteriorly they may reach the *alæ nasi*.

A simple nasal polypus has the appearance of a white grape, rather opalescent, soft to the touch, does not bleed readily, and can usually be moved about by the probe. The examiner distinguishes such from the middle turbinate, which is hard to the touch, either pink or glistening white in colour, and does not move when probed. As this region of the nose is very sensitive, it is better to spray it with a little 2% cocaine solution before palpation is undertaken.

The hypertrophied inferior turbinate may be pale, but is usually red and congested; the firm bone below the thickened mucosa can be felt when touched with a probe. The posterior ends of the inferior turbinates usually show marked hypertrophy.

In chronic posterior sinus suppuration the posterior ends of the middle turbinates show marked hyperplastic changes and also the mucosa of the roof of the posterior choana.

The site of the chronic muco-purulent discharge associated with each sinus involved I have indicated previously under acute nasal sinusitis.

The chronic post-nasal discharge which is often associated with the formation of crusts, causes such conditions as chronic naso-pharyngitis, chronically infected tonsils, hypertrophy of the lymphoid tissues of the lateral pharyngeal bands, chronic laryngitis and bronchitis. The patient complains of phlegm at the back of the nose, soreness of the throat, huskiness of the voice and cough. Not all of these symptoms may be present in each individual case, the cough and post-nasal dropping being most frequently complained of.

Morning headaches and "heaviness" in the frontal region, lack of concentration, poorness of memory and generally feeling tired are often noted.

Proof puncture of a chronically suppurating maxillary antrum usually shows pieces of muco-pus in the return lotion which sink to the bottom of the kidney dish.

Non-Suppurative Chronic Sinusitis.

The non-suppurative chronic sinusitis may be diagnosed from the frequently recurring "colds in the head" that the patient complains of. They are really "sinus colds" and associated with the development of acute exacerbations of the chronically inflamed sinus mucosa. The patient usually states that he has not been in any way exposed to infection and cannot account for having caught the cold.

There is marked general toxæmia associated with these colds which is most pronounced just before the appearance of the discharge. The patient during this prodromal stage often feels very ill. Sometimes there is a rise in temperature and pulse rate, particularly in children. The local signs and symptoms appear with the formation of the discharge and disappear as this clears up. The infected sinus then returns to its non-suppurative state, but it still acts as a focus of infection.

Nasal polypi are not so often present in these cases as in those of the chronic suppurative type.

Proof puncture and lavage of the maxillary antrum in the non-suppurative type, even though marked polypi and hypertrophied mucosa are present in the antrum, yield no muco-pus in the wash-out; the result macroscopically is normal.

X ray films are most useful in the diagnosis of chronic nasal sinusitis. These are taken in different positions in order to bring more clearly into view each individual sinus. When present, hypertrophied mucosa, polypi or fluid can be seen clearly in the film.

The presence of this chronic post-nasal muco-purulent discharge and the toxic absorption from these infected sinuses may give rise to any of the following conditions:

The Respiratory System: Frequent attacks of bronchitis and bronchopneumonia, asthma, bronchiectasis. *The Alimentary System*: Dyspepsia and constipation. *Circulatory System*: Cardiac toxæmia, anæmia. *Nervous System*: Epilepsy, melancholia and various abnormal mental states, neuritis. *Eyes*: Optic neuritis, cyclitis, iritis. *Nose*: Cacosmia or anosmia. *Ears*: Tinnitus, defective hearing. Arthritis. Fibrositis. Dermatitis.

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British Medical Association News.

SCIENTIFIC.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Royal Alexandra Hospital for Children, Camperdown, New South Wales, on August 14, 1930. The meeting took the form of a series of clinical demonstrations by members of the honorary staff.

Cerebellar Tumour.

DR. A. W. CAMPBELL showed a male patient, aged six years, who had been admitted on April 8, 1930. There was nothing of importance in the family history nor in the history of previous illness. For a period of two weeks prior to admission the patient had felt tired, suffered from pains in the head and abdomen, experienced a feeling of weakness of the legs and had been unable to walk steadily. At the time of admission the child appeared to be mentally dull. There was no inequality of the pupils,

which reacted to light. The knee jerks were slightly exaggerated; the plantar reflex was flexor. Kernig's sign was absent. There was no rigidity of the neck, nor was there any head retraction. There was some degree of incoordination. There was no limitation of movement. The child swayed slightly when examined for the presence of Romberg's sign. The gait was ataxic. The blood failed to react to the Wassermann or Kahn test. Radiographical examination, made on April 16, 1930, revealed no actual tumour shadow, but an enlarged skull with fairly thin bones and wide open coronal sutures suggesting increased intracranial pressure. Ataxia and Rombergism became more noticeable. Severe occipital headache developed, attacks of vomiting occurred daily, and there was incontinence of urine and fæces. Between May 20, 1930, and June 3, 1930, the child was given five applications of deep X ray therapy at Saint Vincent's Hospital. On June 28, 1930, the control of micturition and defæcation appeared to be much more efficient and there appeared to be better control over voluntary movements; there were now no headache and no vomiting. The patient became drowsy and recommenced to vomit after further deep X ray applications on July 2 and 3, 1930, but his condition again rapidly improved thereafter. The Honorary Ophthalmologist reported on April 14, 1930, that papillœdema was definitely present on both sides, but on June 16, 1930, he reported the existence of a mild papillœdema on the right side only and some temporal pallor of the disc on the left side, suggestive of partial optic atrophy.

Chronic Lead Poisoning.

Dr. Campbell also showed a female child, aged seven years, a resident of North Queensland, who had been admitted on July 1, 1930. The child had been perfectly well until seven months prior to admission, when she had contracted pertussis. Six months prior to admission she had become subject to attacks of vomiting accompanied by severe headache. Dimness of vision had developed, the attacks of vomiting and headache had become worse, and her gait had become unsteady. The child was stunted. She was remarkably intelligent and able to carry out efficiently all the tests put to her. Her coordination was good. Her pupils were equal and active, her knee jerks could be elicited and she had no Rombergism. There was no staggering or hesitant gait. Sensation and muscular power appeared to be normal. There was no ankle drop or wrist drop. Severe double papillœdema was present. Blood counts were normal and no punctate basophilia was observed. X ray examination revealed a large skull with open sutures suggestive of increased intracranial pressure. There was no reaction to either the Wassermann or the Kahn test. On August 1, 1930, examination of the urine revealed the presence of lead in the definitely pathological proportion of 0·04 milligramme per litre. On August 5, 1930, twenty cubic centimetres of clear fluid under pressure were drawn off by lumbar puncture. On August 6 only two cubic centimetres were obtained, and on August 8 no fluid was obtained. On August 11 a further amount of twenty cubic centimetres under slightly increased pressure was removed.

Congenital Heart Disease.

DR. E. H. M. STEPHEN showed a female child, aged seven years, who had been admitted on July 23, 1930. The child had always been subject to attacks of cyanosis and breathlessness on exertion and had been unable to play or to walk quickly. On the day prior to admission she had had a severe attack which had prostrated her. There was evidence of mental deficiency. The child was cyanosed and her fingers and toes were clubbed. The pulse was regular. The apex beat was diffuse. The most pronounced cardiac murmur was just outside the left border of the sternum in the third intercostal space; from here it was transmitted towards the apical and aortic areas. There was no increase in liver dulness and no tenderness over the liver. There was no polycythæmia. X ray examination revealed an increase in the cardiac shadow to the right and also perhaps at the base of the heart, to the left. The lesion was probably patent *ductus arteriosus* with pulmonary stenosis.

Erythrœdema.

Dr. Stephen also showed a female patient, aged two years and three months, who had been admitted on July 28, 1930, suffering from erythrœdema. The child had been ill for eleven weeks prior to admission. Hands and feet had become red, swollen, peeling and itchy. She had lost appetite and had wasted considerably. The child was very irritable generally, there was excessive sweating, but no photophobia nor coryza. The red blood cells numbered 5,700,000 and the white cells 6,990 per cubic millimetre of blood. The hæmoglobin percentage was 60 and the colour index was 0·7. The polymorphonuclear leucocytes were in the proportion of 36% and the lymphocytes 52%. The quantity of blood calcium was 7·6 milligrammes and of blood phosphorus seven milligrammes per 100 cubic centimetres of blood.

Dr. Stephen also showed two male children, each aged one year, also suffering from erythrœdema. One of these had been ill for five months, weighed 6·93 kilograms (fifteen pounds six ounces) on admission and wasted while in hospital until the weight had dropped to 5·3 kilograms (eleven pounds six ounces).

Bronchiectasis.

Dr. Stephen's next patient was a female child, aged six years, who had been admitted on July 19, 1930. The patient had contracted pneumonia sixteen months prior to admission, and from that time had had a chronic cough and had voided copious quantities of foul-smelling sputum. She had alternately lost and gained weight, had sweated freely at night and had become subject to attacks of breathlessness on exertion. The fingers were clubbed.

Röntgenological examination revealed dulness of maxillary antra and bilateral bronchiectasis. No tubercle bacilli were observed in the sputum.

Cerebro-Spinal Meningitis.

Dr. Stephen's next patient was a female child, aged seven years, who had been admitted on July 31, 1930. Illness had commenced four days prior to admission. Two days later she had become affected with continuous vomiting and spasmodic headache. On the evening of the third day of illness a faint rash had appeared in the groins and on the arms.

When seen on the day of admission she was drowsy and occasionally delirious, but had no vomiting. Her neck was stiff; there was a double Kernig's sign; the knee jerks and plantar reflex were absent. Meningococci were observed in the turbid fluid obtained by lumbar puncture. During eight days 80 cubic centimetres of meningococcal antiserum were administered intrathecally, 30 cubic centimetres intravenously and 130 cubic centimetres intramuscularly. At the time of the meeting the neck rigidity was less obvious and the child seemed more comfortable, though the temperature was still higher than normal.

Dr. Stephen then showed a male child, aged one year and eight months, who had been admitted on July 14, 1930. The patient had been constipated the day prior to admission and on the day of admission had become drowsy and had vomited several times. At the time of admission he had a temperature of 39·01° C. (102·2° F.) and there was some rigidity of the neck. By lumbar puncture twenty-five cubic centimetres of blood-stained fluid under increased pressure were removed. Meningococci were observed in a direct smear of this fluid. On the following day the *cisterna magna* was punctured and twenty cubic centimetres of blood-stained fluid removed. Five cubic centimetres of meningococcal antiserum were injected into the *cisterna*, thirty cubic centimetres intramuscularly and twenty-five cubic centimetres intravenously. During the succeeding six days a further quantity of 130 cubic centimetres of antiserum was administered. The pyrexia subsided on the fifth day of illness and the patient recovered, though convalescence was interfered with somewhat by the advent of a serum rash which lasted for a period of six days and caused a rise of temperature.

Nephrosis.

Dr. Stephen's next patient was a female child, aged one year and four months. Ten days prior to admission

puffiness had appeared round her eyes, and seven days later she had become listless. The day before admission her mother had noticed that the child's legs had become swollen and that the amount of urine had diminished. At the time of admission the face was very puffed, the eyes were almost closed, there was œdema of ankles and legs. The urine contained a large amount of albumin and casts, but no blood. The urea concentration factor was 35. The blood urea nitrogen was 24 milligrammes per 100 cubic centimetres of blood; the urea in the urine amounted to 1·7%. The abdomen was not distended at first, but gradually became more and more tense until eventually the child was unable to sit up. One month after admission she commenced to pass a lot of urine. The œdema then disappeared rapidly and her weight was reduced from 11·88 kilograms (twenty-six pounds six ounces) to 8·91 kilograms (nineteen pounds twelve ounces) in one week. On August 5, 1930, the blood urea nitrogen amounted to 15 milligrammes per 100 cubic centimetres of blood, the urea in the urine amounted to 2·6%, and the urea concentration factor was 86. Albumin was absent from the urine. At the time of the meeting the blood creatinine amounted to 201 milligrammes and the blood cholesterol to 151 milligrammes per 100 cubic centimetres of blood.

Dextrocardia.

DR. MARGARET HARPER showed a male patient, aged five years, who had been admitted on July 15, 1930, suffering from pneumonia. On examination at the time of admission the apex beat was observed to be in the fifth right intercostal space 8·75 centimetres (three and a half inches) from the mid-line. The left border of the heart was 12·5 millimetres (half an inch) lateral to the left border of the sternum. There was nothing abnormal in the heart sounds and no adventitious sounds were audible. There were signs of pneumonic consolidation at the base of the right lung. Radiographical examination revealed a condition of dextrocardia and an area of dulness at the base of the right lung.

Pyelitis and Nephritis.

DR. M. J. PLOMLEY showed a female patient, aged seven years, who had been subject to "kidney trouble" since the age of two years. The child had been passing turbid urine frequently, especially at night. There had been no dysuria. The appetite had been poor and headache frequent. At the time of admission the tonsils were small and the abdomen soft. There was no tenderness over the loins and no masses were palpable. The specific gravity of the urine was 1005; albumin and pus were present. An alkaline mixture was administered over a period of three weeks, and then an acid mixture containing hexamine was given. Fluids were given in profusion and protein was restricted.

Acute Nephritis with Convulsions.

Dr. Plomley's next patient was a female child, aged eight years, who had had a "cold" for two weeks prior to admission and who had commenced to vomit two days before admission. On the morning of admission she had become delirious and had been seized with convulsions which continued for two hours after admission. There was œdema of the face and legs, and urine was scanty and contained albumin and blood. Morphine was administered, bowels and stomach were washed out and hot packs were applied to the loins. Venesection was performed. The patient made a steady recovery and the urine became quite clear.

Meningococcal Meningitis.

Dr. Plomley's next patient was a male infant, aged four months, who had been admitted on June 16, 1930. Before admission the patient had suffered from vomiting for three days, diarrhœa for one day and a slight cough. At the time of admission the fontanelle was bulging. There was slight head retraction, the pupils were equal and active, Kernig's sign was present. Nothing abnormal was detected in the circulatory system or the respiratory system. Lumbar and cisternal punctures were performed. For the first week the condition was unaltered, but improved gradually thereafter. Lumbar puncture was made on alternate days for one month and then at intervals gradually increasing to seven days.

Bone Dystrophy.

Dr. Plomley then showed a female child, aged two years and nine months, who had been in an institution since birth and concerning whose family history nothing was known. At the time of admission the child was suffering from stomatitis and painful joints. The ends of the long bones were enlarged. There was no tenderness. There appeared to be no excess of fluid in the joints. The costo-chondral junctions could be palpated. No abnormality of the skull was detected. The teeth were in good condition, but there was some bleeding from the gums. The child was unable to stand. Nothing of importance was observed in the fauces; the tongue was clean. The abdomen was protuberant, but there was no enlargement of the liver or spleen. Nothing abnormal was detected in the circulatory, respiratory, nervous or genito-urinary system. The child was quite bright mentally. There was no reaction to the Wassermann or the Kahn test. The red blood cells numbered 5,950,000 per cubic millimetre of blood, the colour index was 0·9 and the hæmoglobin percentage was 75. The white cells numbered 11,900 per cubic millimetre; 49% of these were polymorphonuclear leucocytes and 42% were lymphocytes. The radiographical appearances did not suggest rickets. There were periosteal changes in both humeri, but the appearances at the ends of the diaphyses were not suggestive of scurvy. All the bones had a curious transradiance, a somewhat "glossy" appearance, which may be observed in scurvy, pink disease and *fragilitas ossium*. The chondral ends of the ribs were greatly enlarged. The long bones were shorter than normal, but not flared out as in achondroplasia. The skull appeared to be normal. The institution of proper feeding on a full diet which included orange juice and cod liver oil, effected a rapid improvement. Three weeks after admission the child stood and shortly afterwards was able to walk.

Renal Rickets.

DR. C. L. S. MACINTOSH showed a female child who had suffered from increasing weakness and continuous headache, and whose breath had been foul for a period of two weeks prior to admission on February 4, 1930. At the time of admission the child was sallow in appearance and there was some excess of pigmentation. Nothing abnormal was detected in heart or lungs and the spleen was not palpable. There was nothing abnormal noted with regard to the urine. The red blood cells numbered 4,000,000 and the white cells 5,000 per cubic millimetre of blood. The hæmoglobin percentage was 42. Radiographically there were observed in the ends of the diaphyses in the region of the knee and ankle, changes suggestive of renal rickets. In the skull there were some small irregular areas of osteosclerosis typical of early Paget's disease. There was some thickening at the base of the skull. There was no reaction to the Wassermann test. The blood urea nitrogen amounted to 31 milligrammes per 100 cubic centimetres of blood, the blood calcium 6·6 milligrammes and the inorganic phosphorus 7·5 milligrammes. The urea concentration test was normal.

Fibrocystic Disease of Bone.

DR. P. L. HIPSLEY showed two male patients, each aged ten years, who were suffering from fibrocystic disease. In one instance the child had sustained a fracture through a fibrocystic area in the femur one week prior to admission. In the other instance the child had injured his arm while throwing a stone three weeks prior to admission. The diagnosis in each instance was made by radiological examination.

Cardiospasm.

Dr. Hipsley also showed a female child, aged seven years, who had been subject to attacks of vomiting at irregular intervals during a period of six months prior to admission. The period occupied by each attack had varied in length from one week to one month. During the

attacks vomiting occurred after each meal. X ray examination revealed dilatation of the œsophagus with a funnel-shaped narrowing at the lower end.

Fractured Cervical Spine.

DR. F. C. ROGERS showed a male child, aged seven years, who, four days before admission on February 5, 1930, had fallen a distance of 1·5 metres (five feet) on to the back of his head. At the time of admission there were no symptoms save stiffness of the neck. There was no evidence of involvement of the nervous system. Radiological examination revealed a fracture of the arches of the first, second, third and fourth cervical vertebræ with very little displacement. Extension with a weight of 0·9 kilogram (two pounds) was applied on February 14, 1930, and removed on March 11, 1930, when the head was immobilized between sandbags. On June 12, 1930, radiological examination revealed bony union. The sandbags were removed on June 17, 1930.

Ollier's Disease.

DR. WILFRED VICKERS showed a female patient, aged twelve years, who had commenced to limp in February, 1928, and had been admitted to hospital in August of that year. Radiologically a large transradiant area was observed toward the lower aspect of the neck and upper end of the shaft of the femur on the inner surface. The appearance suggested the condition described by Ollier as chondrodysplasia, but the typical small bony masses usually found in such lesions were absent.

A note made on November 25, 1929, was to the effect that there was no advance and if anything the lesion appeared to be contracting, especially at the upper margin. On August 2, 1930, X ray examination revealed a fracture through the transradiant mass.

Spastic Paralysis.

DR. H. G. HUMPHRIES showed a female child, aged twelve years, suffering from a right-sided spastic hemiplegia and strabismus. The spasticity had first been noted when the child had commenced to crawl. The child's mentality was good. On July 30, 1930, lumbar ramisection on the right side was performed, and the right *tendo Achillis* was divided. It was proposed to perform cervical ramisection and tenotomy of *pronator teres* muscle later.

Dr. Humphries also showed a male child, aged four years, suffering from spastic diplegia. Lumbar ramisection and tenotomy of *tendo Achillis* was performed on the left side on June 12, 1930, and on the right side on June 26, 1930.

Pyelitis and Nephritis.

DR. L. A. DEY showed a female patient, aged nine years, whose illness had commenced three years prior to admission. The child had "wet the bed" persistently and the urine had been foul-smelling. There had been no painful micturition nor headache and the appetite had remained fair. At the time of admission the tongue was furred and the abdomen soft. No masses were palpable. There was no tenderness over the kidneys. The urine was acid in reaction and contained albumin and pus. An acid hexamine mixture and copious fluids were administered.

Congenital Abnormality of the Bowel.

DR. KEITH SMITH showed a male patient who had been admitted on July 16, 1930, at the age of forty-four hours. The bowels had not acted since birth. At the time of admission the child was pale, the pulse rather soft and the general condition poor. The abdomen was very distended. A stricture was palpable five centimetres (two inches) from the anus. At an immediate abdominal operation no large bowel was found and ileostomy was therefore performed. On July 19, 1930, the child passed a small constipated stool *per rectum* and several after that date, then, over a period of three weeks, only one. She became gradually more wasted. On July 25, 1930, a barium enema was observed by X rays to pass into the ileum, while barium injected through the ileostomy opening was observed to enter the large bowel. On August 8, 1930, an opaque meal was observed to pass no further along the intestinal tract than the site of ileostomy.

Obituary.

ARTHUR NORRIS WILKINSON.

As announced in the issue of October 4, 1930, Dr. Arthur Norris Wilkinson died at Bendigo, Victoria, on September 17, 1930. He was born at Norwich, England, on April 14, 1871. He was the eldest son of the late Charles Wilkinson, of Offerton Road, Clapham, London, and went to school at the Merchant Tailors' School. He qualified in 1895 and became a licentiate of the Royal College of Physicians, London, and a member of the Royal College of Surgeons of England. After graduation he went to China as a ship's surgeon and settled there. Here he had charge of a Chinese hospital for some years. He filled the position of American Vice-Consul and illustrated "The History of Formosa," a work written by his friend, Mr. Davidson, who was American Consul. He left China on account of his health and settled for a short time in Tasmania. He then moved to Port Pirie, South Australia, where he was attached to the Port Pirie Hospital as an honorary medical officer. Subsequently, for fifteen years he practised at Inglewood, Victoria, and for four years at Yea. Two years before his death he retired from active practice on account of ill health.

Arthur Norris Wilkinson had as his hobbies photography and motor car driving. He worked quietly and conscientiously and made himself necessary to those with whom he came into daily contact.

A fellow practitioner and friend who wishes to remain anonymous, writes of Wilkinson that his interest in both the scientific and human aspects of medical practice never waned. "He had a natural curiosity in the investigation and treatment and saw glamour in the practice of it. His means of livelihood was, fortunately for him and his patients, his great delight—perhaps his only one, for he had no hobbies, no relaxation. Six weeks would probably cover all his holidays in the last twenty years, a concentration which apparently never fatigued him or affected his health. He leaves a record of a life's work, whole-souled and whole-hearted, in which the material side meant little. If the spirit in which we do our work counts for anything, his was a life well spent."

Correspondence.

SPREADING GANGRENOUS INFLAMMATION.

SIR: I read with interest in the journal of September 20 the report by Dr. Poate of his case of spreading gangrenous inflammation. Dr. Poate concludes by remarking that neither himself nor any of his colleagues had seen anything like it before, nor could it be identified with any recognizable pathological process.

I should like to record that in January of this year I treated what I feel sure, from the description and illustration given, was an identical condition, at least as far as clinical manifestations were concerned. The patient in this case was a woman of eighty-one years who was in good health considering her age. She had been accidentally pricked by a rose thorn four days previous to my being called in to see her and in that time had developed around the puncture wound a red brawny swelling resembling, as in Dr. Poate's case, a commencing carbuncle. At the time of the injury the old lady had "squeezed the blood out, washed the puncture and applied spirit." The brawny swelling proceeded to spread in all directions, particularly up the arm, and as time went on the indurated tissues became black and gangrenous and finally sloughed away. The condition after two weeks had reached the axilla and was passing over on to the chest wall, when the patient died of exhaustion and toxæmia. All that was left of the arm after the process had done its work was deep fascia, muscle and bone, with one or two uneroded superficial veins, the muscle surface

being left clean and unaffected. Toxæmia, although marked, did not interfere with cerebral function, the patient remaining mentally alert till the day preceding death. The process could be described adequately as a horrible sloughing away of all tissues down to the deep fascia, which structure was left unaffected, as also were the muscles in the whole extent of the limb.

All treatment was unavailing. In the early stages fomentations and ichthyol glycerine applications had been utilized. Later, continuous baths of eusol, hydrogen peroxide and hypertonic saline were tried. In addition, the patient had received the usual tonic and stimulant treatment, an adequate amount of polyvalent antistreptococcal serum and "Trimine" injections (colloidal manganese, iron and zinc). She was continuously looked after by two special nurses in a private hospital. Amputation was advised, but permission refused on account of the patient's age. At first it was suspected that the condition was the result of the entry of gas-forming bacilli, but as no suspicion of emphysema developed in the affected tissues at any stage, this theory was rejected.

Two or three colleagues who saw the patient, were unable to classify the condition and had seen nothing like it before. A bacteriological examination was not made, nor a vaccine prepared, so that the causal organism will have to remain in doubt. It is reasonable to suppose that a streptococcus was responsible, but why in both cases should it evidence such a remarkable selective affinity for the superficial fascia alone?

Yours, etc.,

GILBERT G. BRADLEY.

Northbridge,
New South Wales.
September 23, 1930.

POST-GRADUATE EDUCATION IN TASMANIA.

SIR: I wish on behalf of the Tasmanian Branch of the Association to draw your attention to a paragraph appearing in the Education Number of THE MEDICAL JOURNAL OF AUSTRALIA.

Under "Post-Graduate Education" it is stated as regards such work in Tasmania: "In the southern part of the Island occasional lectures have been given. Possibly with the reintroduction of the honorary system in the Hobart General Hospital, a more lively interest in clinical medicine and so in post-graduate teaching will become manifest."

It may be of interest to your readers to know that after the splendid post-graduate course and visit of Professor Fraser to Melbourne in 1928, it was resolved to form a committee to organize post-graduate work in Hobart.

Since the beginning of 1929 we have had visits from Mr. Gordon Shaw, Dr. James Bell, Sir Charles Ballance, Dr. Jean Macnamara and Dr. Stewart Cowen. In addition to lectures, operative work, demonstrations of surgical anatomy and clinical medicine have been undertaken.

The attendance at these short week-end courses has been practically 75% or more of the practitioners of Hobart and the south, with men from the north and north-west of this State.

The organization of this work has been carried out in spite of there being no big general hospital to provide cases and material for our distinguished visitors to demonstrate the practical application of their addresses.

The profession here felt a deep sense of obligation to them. Their knowledge and enthusiasm have not only helped us in a period when we were much in need of encouragement, but materially contributed to a settlement of our long-standing hospital dispute.

Yours, etc.,

WILLIAM L. CROWTHER,
For Organizing Committee,
Post-Graduate Work.

Hobart.
October 30, 1930.

Corrigendum.

OUR attention has been drawn to several inaccuracies in the Education Number, published on October 25, 1930.

The subscription to the Queensland Branch of the British Medical Association was wrongly stated. Senior metropolitan members pay £5 10s. *per annum*, senior country members pay £4 10s., junior members (under three years' qualification) pay £2 12s. 6d., metropolitan professional officers (senior members) pay £4 10s., country professional officers (senior members) pay £3 10s. Metropolitan members are those resident within the Greater Brisbane area. Professional officers include whole-time officers of the public services.

In regard to the Department of Defence, the Director-General Medical Services is not allowed private practice. There is now no Director of the Royal Australian Air Force. This service is administered and controlled by the Director-General Medical Services through his representative in the several States, the Deputy Director Medical Services (Air) who is a part-time Citizen Force officer. There are two Citizen Force medical officers in New South Wales and a Deputy Assistant Director Medical Services (Air) on the staff of the Deputy Director Medical Services, Second Military District.

The arrangements in connexion with school medical inspection in Western Australia are not as stated. There are three full-time medical officers, three school nurses and three full-time dentists. In the opinion of Dr. Everitt Atkinson the staff is adequate and satisfactory work is being carried out. Dr. Roberta Jull is no longer in the service.

Proceedings of the Australian Medical Boards.

NEW SOUTH WALES.

THE undermentioned have been registered under the provisions of the *Medical Act*, 1912 *and* 1915, of New South Wales, as duly qualified medical practitioners:

Ginsburg-Lapin, Mollie Amelia, M.B., B.S., 1930 (Univ. Sydney), 5, Malvern Court, Croydon Avenue, Croydon.

Gordon, Charles Patrick, M.B., B.S., 1930 (Univ. Sydney), 259, Walker Street, North Sydney.

Heighway, Freida Ruth, M.B., B.S., 1930 (Univ. Sydney), 7, Ethel Street, Burwood.

Hewitt, Lance Ellis, M.B., B.S., 1930 (Univ. Sydney), Wesley College, Newtown.

Hobson, Arthur Francis, M.B., B.S., 1930 (Univ. Sydney), 10, Jersey Road, Strathfield.

Horsley, Charles Houston, M.B., B.S., 1930 (Univ. Sydney), 12, Edgeware Road, Enmore.

Huntley, Ralph Clifford, M.B., B.S., 1930 (Univ. Sydney), 41, Francis Street, Kogarah.

Kelleher, Frederick, M.B., B.S., 1930 (Univ. Sydney), 16, Kingston Street, Haberfield.

Meares, Stanley Devenish, M.B., 1930 (Univ. Sydney), 6, Chandos Street, Ashfield.

Melville, Hugh James, M.B., Ch.B., 1929 (Univ. Edinburgh), 80, Roseville Avenue, Roseville.

McCarthy, Charles Maurice, M.B., B.S., 1930 (Univ. Sydney), 28, Middleton Street, Stanmore.

McCormack, Patrick Edward, M.B., B.S., 1930 (Univ. Sydney), 24, Lamrock Avenue, Bondi.

McGrath, Walter Stafford, M.B., B.S., 1930 (Univ. Sydney), 2, Dutruc Street, Randwick.

McIlrath, Muriel Betty, M.B., B.S., 1930 (Univ. Sydney), "Ingleholme," Boomerang Street, Turramurra.

MacKellar, Duncan Gordon, M.B., B.S., 1930 (Univ. Sydney), 70, Piper Street, Leichhardt.

MacMahon, Christine Helen Mary, M.B., B.S., 1930 (Univ. Sydney), 83, Waratah Street, Haberfield.

MacMahon, Dora Kathleen, M.B., B.S., 1930 (Univ. Sydney), Hampden Avenue, Darling Point.
Nelson, Selwyn Graham, M.B., B.S., 1930 (Univ. Sydney), 103, Villiers Street, Rockdale.
Newton-Tabrett, Alice Eileen, M.B., B.S., 1930 (Univ. Sydney), 2, Gordon Road, Roseville.
Quilter, John William, M.B., B.S., 1930 (Univ. Sydney), 29, Darley Road, Randwick.
Rawle, Ronald Maxwell, M.B., B.S., 1930 (Univ. Sydney), 93, New South Head Road, Vaucluse.
Roberts, Charles Eric Elphingstone, M.B., 1930 (Univ. Sydney), 28, Middleton Street, Stanmore.
Ross, Alexander William, M.B., B.S., 1930 (Univ. Sydney), Malton Street, Beecroft.
Rowohl, Willa, M.B., B.S., 1930 (Univ. Sydney), 8, Smith Street, Manly.
Saxby, Noel Hunter West, M.B., B.S., 1930 (Univ. Sydney), 12, Upper Cliff Road, Northwood.
Schafer, Noel Theodore Hannaford, M.R.C.S. (England), 1927, L.R.C.P. (London), 1927, M.B., B.S., 1930 (Univ. Melbourne), c.o. National Bank of Australasia, George Street, Sydney.
Shorter, Alan Ashton, M.B., 1930 (Univ. Sydney), 133, Middle Harbour Road, Lindfield.
Smith, Leslie, M.B., B.S., 1929 (Univ. Melbourne), 15, Kulgoa Road, Bellevue Hill.
Spencer, Stanley Livingstone, M.B., B.S., 1930 (Univ. Sydney), 46, Archer Street, Chatswood.
Starr, Kenneth William, M.B., B.S., 1930 (Univ. Sydney), 121, Cavendish Street, Stanmore.
Tremayne, James Henry Russell, M.B., 1930 (Univ. Sydney), Edina, Stanthorpe, Queensland.
Ward, John Joseph, M.B., B.S., 1930 (Univ. Sydney), 36, Oxford Street, Newtown.
Warden, Douglas Arthur, M.B., 1930 (Univ. Sydney), "Tabourie," Muston Street, Mosman.
Wearn, Albert Arthur, M.B., B.S., 1930 (Univ. Sydney), 774, Gordon Road, Chatswood.
Weaver, Robert Walmer, M.B., B.S., 1928 (Univ. Melbourne), Women's Hospital, Crown Street, Sydney.
Wright, Reginald George, M.B., B.S., 1930 (Univ. Sydney), Frome Street, Moree.
Yeates, Roderick Angus MacRae, M.B., B.S., 1930 (Univ. Sydney), "Rose Bank," Hill Street, Toowoomba.
Yeates, Ronald Herbert, M.B., 1930 (Univ. Sydney), 7, Ayr Street, Ashbury.

Diary for the Month.

Nov. 18.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
Nov. 25.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Nov. 26.—Victorian Branch, B.M.A.: Council.
Nov. 27.—New South Wales Branch, B.M.A.: Branch.
Nov. 27.—South Australian Branch, B.M.A.: Branch.
Nov. 28.—Queensland Branch, B.M.A.: Council.
Dec. 2.—New South Wales Branch, B.M.A.: Organization and Science Committee.
Dec. 2.—New South Wales Branch, B.M.A.: Post-Graduate Work Committee.
Dec. 2.—New South Wales Branch, B.M.A.: Hospitals Committee.
Dec. 3.—Victorian Branch, B.M.A.: Annual General Meeting.
Dec. 4.—South Australian Branch, B.M.A.: Council.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xvi.

BURREN JUNCTION MEDICAL COMMITTEE, NEW SOUTH WALES: Subsidised Doctor.
CHILDREN'S HOSPITAL, INCORPORATED, PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officers.
HOBART PUBLIC HOSPITAL, HOBART, TASMANIA: Junior Resident Medical Officer.
MATER MISERICORDIÆ PUBLIC HOSPITAL, BRISBANE, QUEENSLAND: Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members desiring to accept appointment in ANY COUNTRY HOSPITAL, are advised to submit a copy of their agreement to the Council before signing, in their own interests. Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Hospital. Mount Isa Mines.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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